A Dissertation on

DEXMEDETOMIDINE FOR LAPAROSCOPIC CHOLECYSTECTOMY – COMPARISON BETWEEN TWO DOSES ON THE SYMPATHOADRENAL RESPONSE AND ANAESTHETIC REQUIREMENTS

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DECLARATION BY THE CANDIDATE

I, **Dr. R.PRANEETH,** solemnly declare that the dissertation, titled **DEXMEDETOMIDINE FOR LAPAROSCOPIC CHOLECYSTECTOMY – COMPARISON BETWEEN TWO DOSES ON THE SYMPATHOADRENAL RESPONSE AND ANAESTHETIC REQUIREMENTS**, is a bonafide work done by me during the period of NOVEMBER 2013 to SEPTEMBER 2014 at Government Stanley Medical College and Hospital, Chennai under the expert supervision of **Dr. KUMUDHA LINGARAJ, M.D., D.A.,** Professor, Department Of Anaesthesiology, Government Stanley Medical College, Chennai.

This thesis is submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the rules and regulations for the M.D. degree examinations in Anaesthesiology to be held in April 2015.

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ABBREVIATIONS

CO_2	-	Carbon dioxide
DBP	-	Diastolic Blood Pressure
ECG	-	Electrocardiography
$EtCO_2$	-	End Tidal Carbon Dioxide
FDA	-	Food and Drug Administration
HR	-	Heart rate
IAP	-	Intra-abdominal Pressure
MAP	-	Mean Arterial Pressure
NIBP	-	Non Invasive Blood Pressure
$PaCO_2$	-	Partial Pressure of Carbon Dioxide in Arterial System
PEEP	-	Positive End Expiratory Pressure
$PetCO_2$	-	Post apnoeic End Tidal Carbon Dioxide Pressure
SBP	-	Systolic Blood Pressure
SpO_2	-	Oxygen Saturation
VCO_2	-	Rate of Elimination of Carbon Dioxide

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ABSTRACT

Background: Dexmedetomidine is a selective α2 adrenergic agonist that attenuates the hemodynamic stress response to tracheal intubation and extubation, decreases plasma catecholamine concentrations during surgery and anaesthesia and decreases the perioperative requirements of anaesthetics and analgesics. Laparoscopic surgeries involve unique hemodynamic alterations and Dexmedetomidine may serve the role of an ideal pharmacological adjuvant to obtund these hemodynamic changes during laparoscopic surgeries.

Objectives: The aim of the study was

(i) To evaluate and compare the analgesic and anaesthetic sparing properties of two doses of Dexmedetomidine - 0.2 µg/kg/hr and 0.6 µg/kg/hr

(ii) To study the hemodynamic stabilizing properties of two doses of Dexmedetomidine- 0.2 µg/kg/hr and 0.6 µg/kg/hr

(iii) To study the side effect profile of Dexmedetomidine (bradycardia, hypotension and increased sedation) for laparoscopic cholecystectomy.

Methodology: Sixty ASA class I patients aged 18 – 50 years were randomized into two groups: A and B to receive Dexmedetomidine infusion at 0.2 µg/kg/hr and 0.6 µg/kg/hr respectively , started 10 minutes before induction of anaesthesia. Inj. Fentanyl at a dose of 1µg/kg was administered before induction of anaesthesia. Anaesthesia

was induced with propofol 2 mg / kg IV and muscle relaxation was obtained by Inj. Atracurium at a dose of 0.5 mg/kg. Desflurane was administered at an inspiratory concentration of 3%. Anaesthesia was initially maintained with desflurane at 3 % inspired concentrations with nitrous oxide (3 l / min), oxygen (1.5 l /min) mixture. Intra –operative HR, SBP, DBP, MAP, SpO2, EtCO2 were recorded every 5 min for first 30 min and subsequently every 10 min intervals till the discontinuation of anaesthetic drugs. MAP and heart rate values were maintained within 25 % of baseline values by administering fentanyl boluses of 0.5 ug / kg (up to a maximum of 2 ug / kg) and by varying inspiratory desflurane concentrations. Total fentanyl requirement, average inspiratory desflurane concentrations, HR, SBP, DBP, SPO2, ETCO2, post operative sedation score and intraoperative need for adjuvant such as propofol were studied.

Results: The two groups showed a statistically significant difference in total fentanyl requirements and average inspiratory desflurane concentrations. The requirement of fentanyl was reduced by 20% in the Dexmedetomidine 0.6 group compared to Dexmedetomidine 0.2 group in our study. The average inspiratory concentration desflurane required in group A (Dexmedetomidine 0.2 µg/kg/hr) was 2.66 and the average inspiratory concentration required in group B (Dexmedetomidine 0.6 µg/kg/hr) was 2.57. Stress response to endotracheal intubation resulted in an increase in heart rate and MAP values in both the groups though the degree of increase was lesser in Dexmedetomidine 0.6 group when compared to Dexmedetomidine 0.2 group. But the values remained

within 25 % of the baseline values and the increase was found not to be statistically significant. Both the groups did not encounter a significant increase in heart rate and MAP values on peritoneal insufflations. The number of hypotensive episodes encountered in Dexmedetomidine 0.6 µg/kg/hr group were found to be significantly higher when compared to the Dexmedetomidine 0.2 µg/kg/hr group, though these episodes were transient and could be corrected by a decrease in inspired desflurane concentration and administration of a fluid bolus. The number of hypertensive episodes in Dexmedetomidine 0.2 µg/kg/hr group was significantly higher when compared to Dexmedetomidine 0.6 µg/kg/hr group and all episodes resolved with fentanyl supplementation. Dexmedetomidine at 0.6 µg/kg/hr resulted in a progressive increase in sedation scores and the time to eye opening when compared to Dexmedetomidine at 0.2 µg/kg/hr.

Conclusion: From this study it was concluded that Dexmedetomidine at an infusion rate of 0.6 µg/kg/hr has a better analgesic and anaesthetic sparing property and a better hemodynamic stabilizing property when compared to an infusion rate of 0.2 µg/kg/hr, with increased episodes of transient hypotension, increased postoperative sedation score, with no serious side effects or adverse reactions.

Keywords: Laparoscopy, dexmedetomidine, fentanyl, desflurane, hypertension, hypotension, tachycardia, bradycardia, sedation,

CHAPTER 1

INTRODUCTION

This is a modern era of surgery which has witnessed many new and innovative approaches encompassing minimal intervention. Laparoscopic surgery occupies the centre stage in this modern era. Anaesthesiologist of the modern era has to be trained enough to handle the repercussions of laparoscopy during the perioperative period, permitting safe and effective patient management while undergoing laparoscopic surgeries.

Anaesthesiologist's major concern is to maintain hemodynamic stability during the course of peri-operative period. Minimally invasive surgical procedures have seen a remarkable growth in the last decade. They aim to lessen the trauma of intervention procedures, decrease the postoperative pain, decrease the duration of stay in hospital, hasten the return to normal activities and being cost effective, but still capable of achieving the desired therapeutic result. The first successful laparoscopic cholecystectomy was performed in 1987 and since then emerged rapidly as a technique that effectively substituted traditional open approach to cholecystectomy for symptomatic cholelithiasis.

Laparoscopic surgeries require intraperitoneal insufflations of CO_2 which is associated with significant and unique hemodynamic

alterations such as reduced stroke volume, elevated blood pressures, and increase in systemic and pulmonary vascular resistance. There is only a slight change in the heart rate[1].Problems occurring during laparoscopic techniques are mainly due to combined effects of pneumoperitoneum with carbon dioxide insufflation and patient positioning , which causes several hemodynamic and ventilatory changes.

Anaesthesia for laparoscopic cholecystectomy has traditionally laid emphasis on the use of a variety of pharmacological agents in different combinations for the purpose of maintenance of hemodynamic stability. α_2-adrenergic agonists may serve the role of ideal pharmacological adjuvant for laparoscopic cholecystectomy, as they provide sedation, anxiolysis, hypnosis, analgesia, and sympatholysis[2]. Dexmedetomidine is a highly selective α_2-adrenergic agonist with 1600 times greater selectivity for the α_2 adrenoceptor compared with the α_1 receptor. Dexmedetomidine attenuates the hemodynamic responses to tracheal intubation and perioperative stress, decreases plasma catecholamine concentrations during surgery and anaesthesia and decreases the perioperative requirements of anaesthetics and analgesics.

Various studies have been published, evaluating Dexmedetomidine administered in various bolus doses for premedication. However, studies evaluating the effects of Dexmedetomidine in different dose ranges are essential for the

administration of the drug as a continuous infusion during surgery. Because Dexmedetomidine has a high propensity to produce hypotension and/or bradycardia , various studies are being conducted worldwide to determine an ideal infusion rate for Dexmedetomidine that would maximize the anaesthetic and analgesic sparing effects , while at the same time limiting the occurrence of adverse cardiac effects. This study is aimed to compare and evaluate the efficacy of Dexmedetomidine administered in two different doses, in providing analgesic and anaesthetic sparing effects and in maintaining hemodynamic stability during laparoscopic cholecystectomy.

CHAPTER 2

AIM

To evaluate the analgesic and anaesthetic sparing, and hemodynamic stabilizing properties of two different doses of Dexmedetomidine - 0.2 µg/kg/hr and 0.6 µg/kg/hr for laparoscopic cholecystectomy.

OBJECTIVES

PRIMARY OBJECTIVE

1. To compare the requirement of total opioid (fentanyl) and volatile anaesthetic (desflurane).

SECONDARY OBJECTIVES

1. To compare the hemodynamic parameters (SBP, DBP, MAP, HR)
2. To study the side effect profile of Dexmedetomidine (hypotension, bradycardia and increased sedation).

HISTORY

It was at the beginning of 20th century that the laparoscopic surgery was first introduced by Dimitri Ott, George Kelling, and Hans Christian Jacobeus. Ott examined the peritoneal cavity of pregnant women in 1901, after which a procedure called “koelioscopie “was performed by George Kelling. In the same year a report called “Laparothorakoskopie” was published by Jacobeus. Several European and American authors performed diagnostic laparoscopic procedures in the following years. With the introduction of cold light fiber-glass illumination and rod-lens optical system, laparoscopic techniques became popular among gynecologists. During this period, laparoscopic technique in the field of general surgery was mainly used for diagnostic procedures such as diagnosing hepatic disorders and abdominal trauma until 1983, when the foresight of two surgeons, Lukichev and Muhe lead them to successfully perform the first laparoscopic cholecystectomy. They did not receive much attention for their accomplishment and general surgeons became greatly interested in laparoscopic surgeries only after the French gynecologist Mouret performed the first laparoscopic cholecystectomy in 1987 using four trochars. Laparoscopic surgery has undergone tremendous advancement over past two decades[3].

The α_2-adrenergic agonists provide sedation, anxiolysis, hypnosis, analgesia, and sympatholysis. The initial stimulus for the use of α_2

agonists erupted from the observations made with the use of clonidine. Dexmedetomidine is a highly selective α_2 agonist with 1600 times greater selectivity for α_2 receptors compared with the α_1 receptors. FDA in 1999 approved Dexmedetomidine only as a short term sedative for mechanically ventilated ICU patients (< 24 hours). The drug is now being used outside the ICU in various settings, including sedation and adjunct analgesia in the operating room, sedation in various diagnostic procedures, and for attenuation of withdrawal and detoxification[2].

LAPAROSCOPIC SURGERY

Laparoscopic surgeries involve the creation of pneumoperitoneum and various patient positions that induce pathophysiological changes, which in turn complicate anaesthetic management. Laparoscopic surgeries result in alterations in ventilator and respiratory mechanics, produce various hemodynamic alterations and also cause diverse physiological disturbances due to patient positioning.

ANAESTHETIC CONSIDERATIONS OF LAPAROSCOPY

(i) VENTILATORY AND RESPIRATORY CHANGES DURING LAPAROSCOPY

The creation of pneumoperitoneum results in four principle respiratory disturbances – CO_2 subcutaneous emphysema, pneumothorax, gas embolism and endobronchial intubation[4].

Pneumoperitoneum leads to a decrease in thoracopulmonary compliance, decrease in functional residual capacity, elevates the diaphragm and results in atelectasis and causes ventilation – perfusion mismatch[5, 6, 7]. $PaCO_2$ progressively increases on the creation of CO_2 pneumoperitoneum and reaches a plateau after 15 – 30 minutes of CO_2 insufflation. The causes are multifactorial including CO_2 absorption across the peritoneal layers, mechanical causes such as abdominal distension by CO_2 pneumoperitoneum, patient positioning and mechanical ventilation resulting in ventilation perfusion mismatch. $PaCO_2$ should be maintained within the physiological range by altering the mechanical ventilation.

CO_2 Subcutaneous Emphysema

CO_2 subcutaneous emphysema results from accidental extraperitoneal insufflation of CO_2. This condition results in the increase of VCO_2, $PaCO_2$, and $PetCO_2$ [8]. CO_2 subcutaneous emphysema results in a progressively increasing $PetCO_2$ that occurs after the $PetCO_2$ value has plateaued. The degree of increase in VCO_2 makes it impossible to prevent hypercapnia by adjustment of ventilation. Under these circumstances the procedure is temporarily discontinued and recommenced after the normalisation of $PaCO_2$ using lower insufflation pressures. Mechanical ventilation should be continued till the correction of hypercapnia to prevent increases in work of breathing.

Pneumothorax, Pneumomediastinum, Pneumopericardium

Pneumoperitoneum may result in the diffusion of gas leading to pneumomediastinum, pneumothorax and pneumopericardium[9,10]. These conditions result in respiratory and hemodynamic disturbances. Capnothorax leads to increased airway pressures and reduced thoracopulmonary compliance. VCO_2, $PaCO_2$, and $PetCO_2$ all increase from their baseline values. Alveolar rupture leading to secondary pneumothorax results in decreased $PetCO_2$ because of decreased cardiac output. Fall in oxygen saturation and hemodynamic changes should raise the suspicion of tension pneumothorax. Pneumothorax that occurs in the absence of pulmonary trauma, by a highly diffusible gas such as CO_2 resolves spontaneously without thoracocentesis. Capnothorax occurring during laparoscopy can be treated by the application of PEEP, rather than a chest tube placement[11] .Pneumothorax that results from rupture of bullae, should be treated by thoracocentesis.

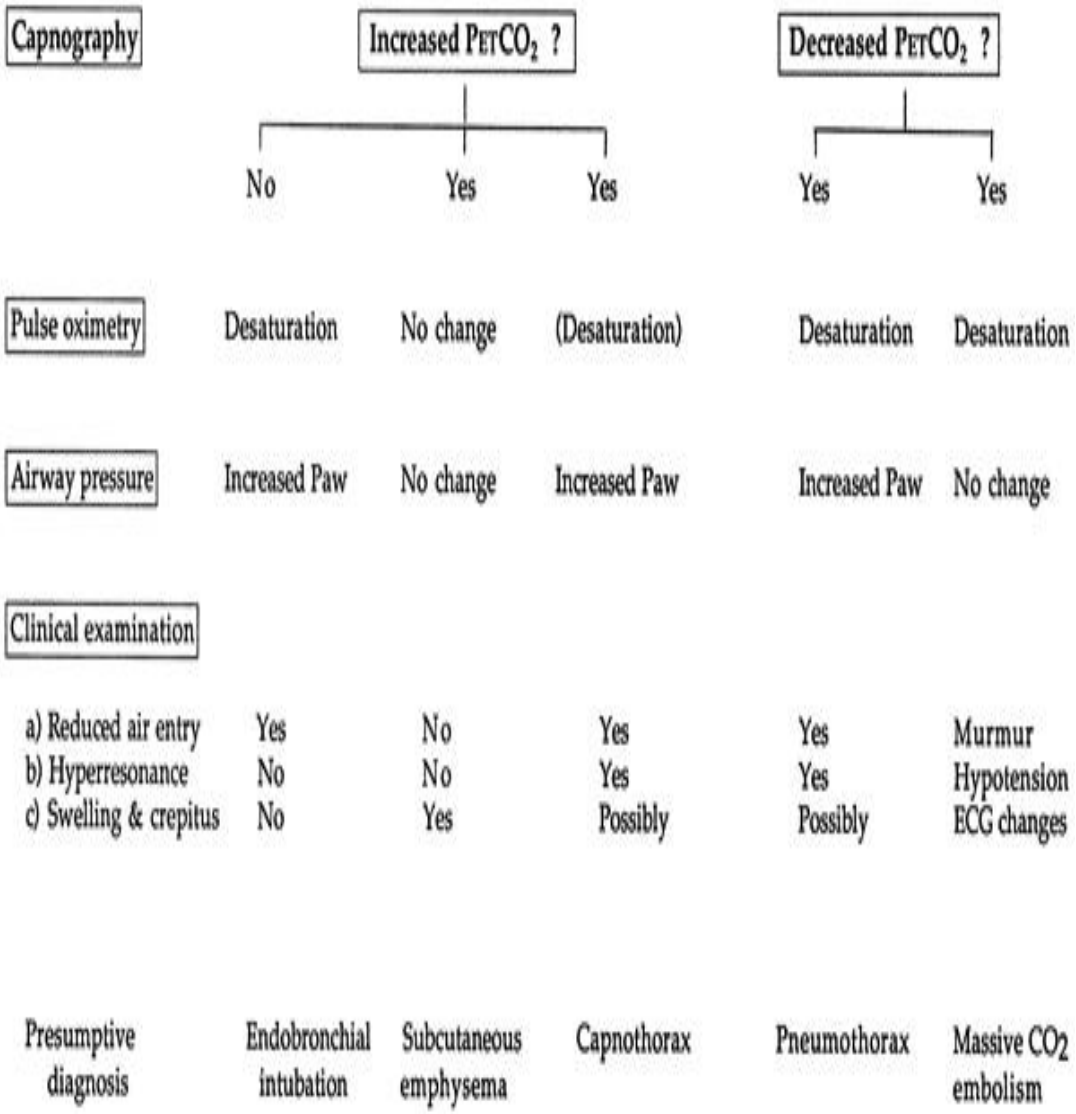


Figure 1: Diagnosis of respiratory complications during laparoscopy. ECG, electrocardiographic; Paw, airway pressure; Petco2, end-tidal carbon dioxide tension.[12]

Endobronchial Intubation

Pneumoperitoneum causes the diaphragm to move cephalad which in turn results in cephalad displacement of carina leading to a potential serious chance of endobronchial intubation. This causes a fall in oxygen saturation and an increase in airway pressures[13].

Gas Embolism

Gas embolism by CO_2 may result from direct needle puncture into a blood vessel or trochar placement or direct insufflation into an abdominal organ. High pressure insufflation of gas at a rapid rate results in "gas lock" right atrium and vena cava, leading to a decrease in venous return and cardiac output and resulting in circulatory collapse. Gas embolism results in a sudden fall in $PetCO_2$ due to decrease in cardiac output and the resulting enlargement of the physiologic dead space. Treatment revolves around immediate release of pneumoperitoneum after the cessation of CO_2 insufflation. Patient is placed in Durant (head down and left lateral) position. Hyperventilation may result in CO_2 excretion. Central venous catheter can be introduced and used for aspiration of gas. Cardiopulmonary resuscitation becomes necessary in cases of massive embolism and should be initiated early. Hyperbaric oxygen treatment and cardiopulmonary bypass are used to treat massive gas embolism[14].

(ii) HEMODYNAMIC COMPLICATIONS OF LAPAROSCOPIC SURGERY

Alterations in hemodynamics are due to changes resulting from the varied effects of CO_2 pneumoperitoneum, positioning of the patient, anaesthesia and hypercapnia. Hemodynamic changes are characterized

by decreased venous return and cardiac output, increase in arterial pressures and peripheral vascular resistance.

Cardiac output decreases due to multifactorial causes. Venous return is characterized by a transient increase at low IAPs (< 10 mm Hg), followed by a decrease[15]. Progressively increasing IAPs results in caval compression, venous pooling and increased venous resistance[15,16]. Decreases in venous return result in a decreased left ventricular end diastolic volume. However, cardiac filling pressures are paradoxically increased by pneumoperitoneum due to increased intrathoracic pressure. IAPs of up to 15 mm Hg do not cause a significant decrease in ejection fraction of left ventricle. Pneumoperitoneum and increasing IAPs result in an increase in peripheral vascular resistance and this is not a reflex sympathetic response to decreased cardiac output[17]. This increased afterload resulting from pneumoperitoneum and increased IAP may prove detrimental in patients with compromised cardiac status.

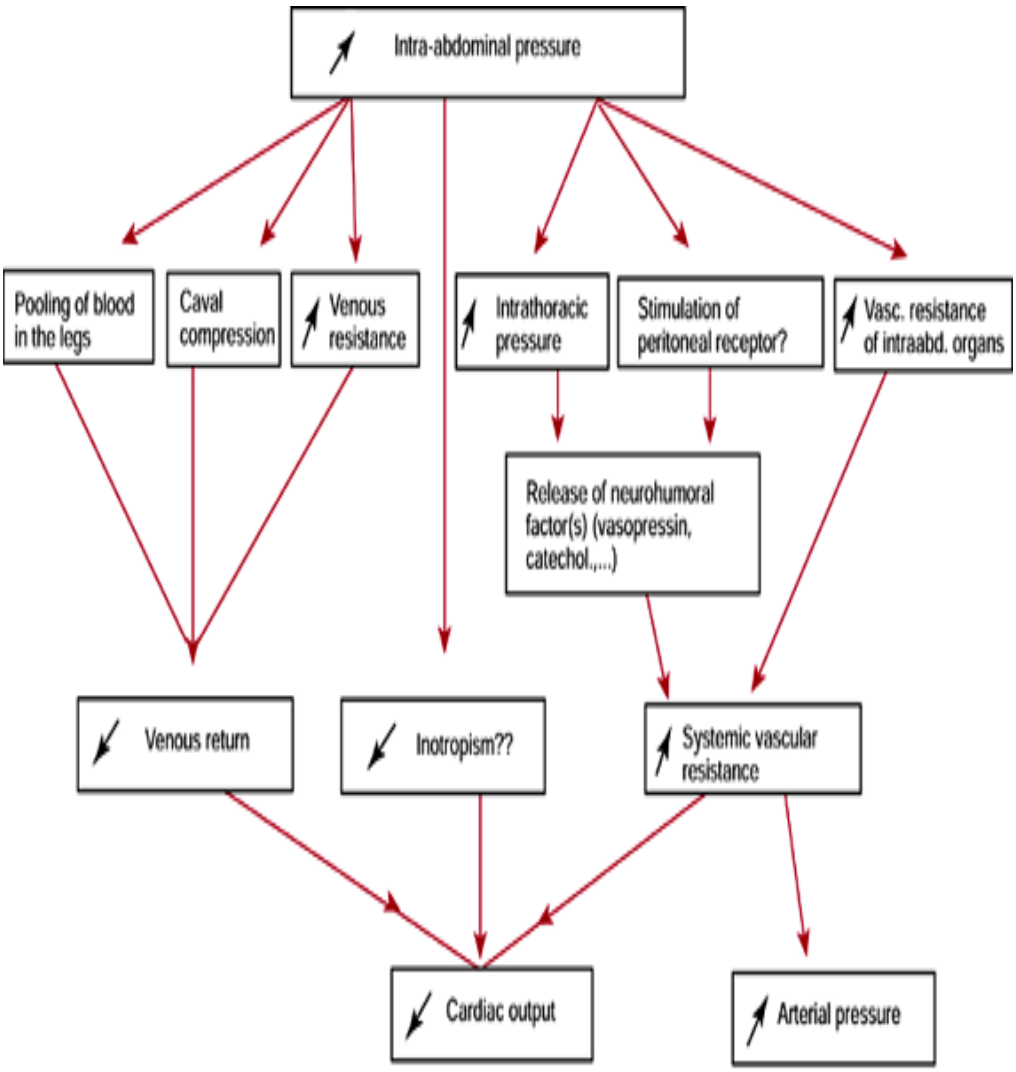


Figure 2: Schematic representation of CO_2 pneumoperitoneum resulting in decreased cardiac output by various mechanisms

Patient position also significantly alters the systemic vascular resistance, with Trendelenberg position attenuating the increase, and head –up position aggravating the increase. The increase in peripheral vascular resistance is probably mediated by both mechanical and neurohumoral factors[18].

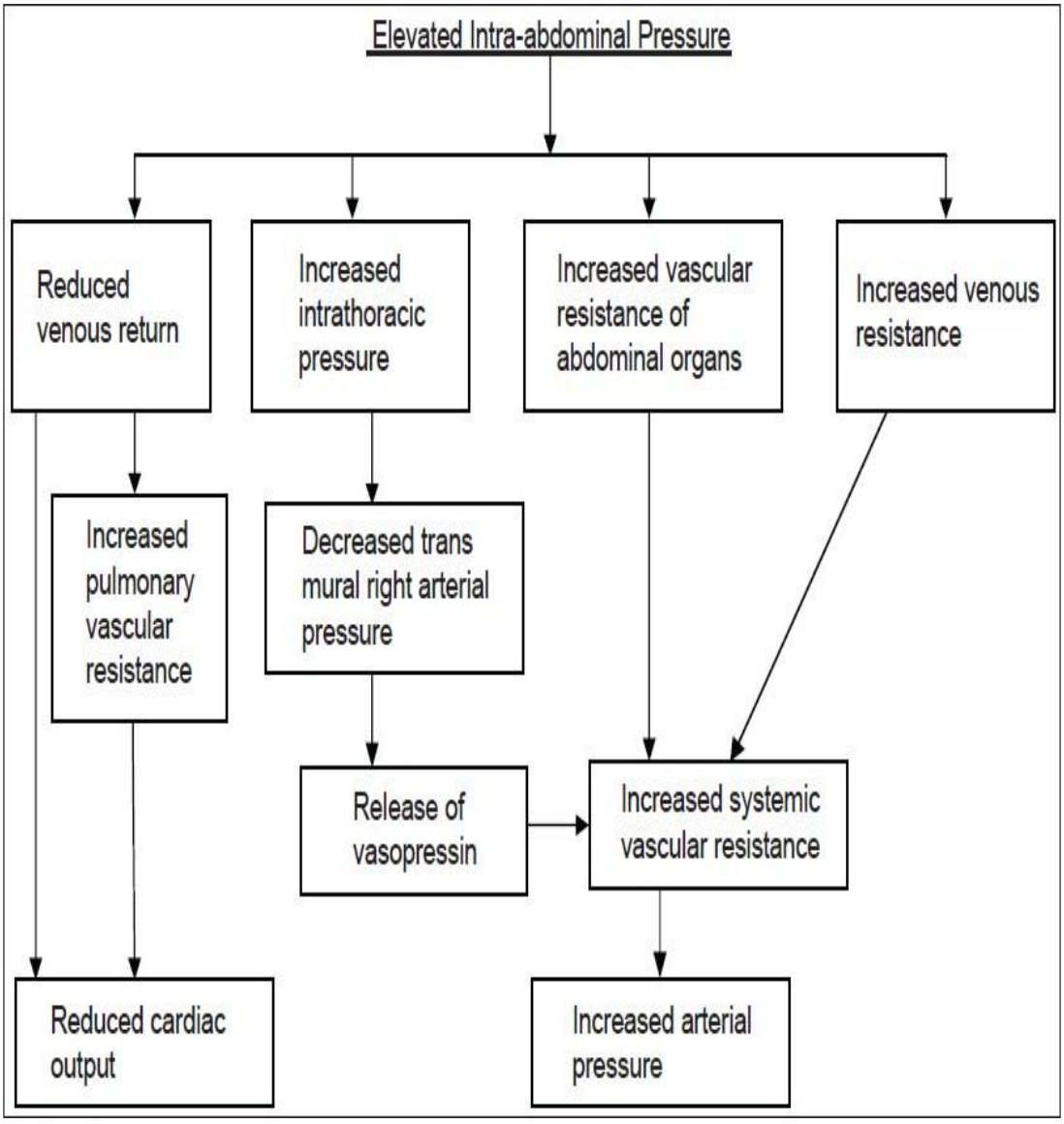


Figure 3: Effects of elevated intra-abdominal pressure

Catecholamines, the renin-angiotensin system, and specifically vasopressin are released during the presence of pneumoperitoneum, which may contribute to increase in afterload. Mechanical stimulation of peritoneal receptors also results in vasopressin release, increased systemic vascular resistance and increased arterial pressures[19].

(iii) EFFECTS OF PNEUMOPERITONEUM ON REGIONAL HEMODYNAMICS

Increased intraabdominal pressure and head –up position results in venous stasis in lower limbs, predisposing to the development of thromboembolic complications[20]. Renal function progressively deteriorates with a fall in urine output, glomerular filtration rate and renal plasma flow, with increases in intraabdominal pressure[21]. Cerebral blood flow velocity increases with CO_2 insufflation. But with the maintenance of normocarbia, pneumoperitoneum along with the head – down position does not induce harmful alterations in intracranial hemodynamics[22].

(iv) ARRHYTHMIAS DURING LAPAROSCOPY

Cardiac arrhythmias occur due to several reasons during laparoscopy. CO_2 insufflation causes stretching of peritoneum, leading to reflex increase in vagal tone which produces bradycardia, cardiac arrhythmias and asystole[23]. This reflex vagal stimulation is accentuated in patients taking β-blockers and in inadequate planes of anaesthesia. The reflex vagal stimulation is treated by interrupting the insufflations of CO_2, intravenous atropine administration and by deepening the plane of anaesthesia after normalization of heart rate.

Cardiac arrhythmias most often occur during the early stages of insufflations, when the hemodynamic disturbances due to CO_2 insufflation are the most profound. In patients with latent cardiac disease, arrhythmias also suggest an intolerance to these hemodynamic changes .Gas embolism is another cause for cardiac arrhythmias.

(v) IMPLICATIONS OF PATIENT POSITIONING DURING LAPAROSCOPY

Patient positioning is determined by the site of surgery. Pelvic and lower abdominal surgeries are performed with a head down tilt. Head up tilt is preferred for upper abdominal surgeries. These variations in patient positions contribute to various pathophysiological changes that occur during laparoscopy.

Cardiovascular Effects

Trendelenberg position results in increased central venous pressure and cardiac output. Baroreceptor reflex response activation leads to systemic vasodilatation and bradycardia .General anaesthesia usually attenuates these reflex responses in hemodynamics, and the hemodynamic alterations induced by laparoscopy in this position are usually not significant in normotensive subjects[24]. But these alterations are accentuated in patients with ischemic heart disease leading to increased myocardial oxygen demand. Trendelenberg position also

causes raised intracranial pressures in cases of decreased intracranial compliance. Head-down position increases intravascular pressures in the upper torso, decreases blood pressures in the lower abdominal and pelvic viscera, thereby decreasing the blood loss due to decreased transmural pressures but increasing the risk of gas embolism[25].

Head up position leads to decreased cardiac output and mean arterial pressures, due to decreased venous return[26].

Lithotomy position leads to accentuation of the venous stasis that occurs in the head up position.

Respiratory Changes

Head down position leads to a decrease in functional residual capacity, total lung volumes and pulmonary compliance as well as causes basal atelectasis. These changes are accentuated in obese and elderly patients. Head up position is considered to be a more favourable position for respiratory mechanics.

Nerve Injury

Nerve compression is a common complication occurring in all positions. Arm should not be abducted beyond 90 degrees. While placing shoulder braces for head down position, great caution must be exercised so that the brace does not impinge on the brachial plexus. Lithotomy position results in injury to the common peroneal nerve and

should be protected .Laparoscopic surgeries for prolonged periods in lithotomy position may result in lower limb compartment syndromes.

(vi) BENEFITS AND CONSEQUENCES OF LAPAROSCOPIC CHOLECYSTECTOMY

Multiple intraoperative derangements caused by pneumoperitoneum are counterbalanced by postoperative benefits including more rapid recovery, heightened feeling of well – being and reduced postoperative fatigue.

(a) Stress Response

Laparoscopic approach to cholecystectomy results in attenuation of acute phase reaction, leading to reduced levels of c – reactive protein (CRP) and interleukin -6 (IL-6) when compared to open technique[27]. Laparoscopic technique also results in a decreased metabolic response (hyperglycaemia, leukocytosis), hence leading to an improved immune function and nitrogen balance[28]. Prolonged exposure and manipulation of intestines and peritoneal incision and trauma are avoided by laparoscopic approach to cholecystectomy, thereby reducing postoperative ileus and fasting, duration of intravenous infusion and hospital stay[29].

Endocrine response to surgery does not differ significantly between laparoscopic and open techniques for cholecystectomy ,

resulting in similar levels of plasma and urinary concentrations of catecholamines and their metabolites in both the techniques[30].Peritoneal stretching leading to pain and discomfort, hemodynamic alterations and ventilator changes caused by CO_2 insufflation may contribute to the stress response induced by laparoscopy, which can be reduced by perioperative administration of α_2 agonists[31].

(b) Postoperative Pain

Pain intensity may be significant after laparoscopic surgeries, with patients complaining of both visceral and parietal pain. Pain after laparoscopy is multifactorial and treated by various methods. Intraperitoneal local anaesthetics are beneficial especially after gynaecological laparoscopic surgeries. CO_2 should be carefully evacuated, as residual CO_2 pneumoperitoneum contributes to postoperative pain[31]. Preoperative NSAIDs, dexamethasone and opioid administration have all contributed to decreased pain intensity after laparoscopic surgeries. Multimodal analgesia should be provided to attenuate laparoscopy induced pain.

(c) Postoperative Pulmonary Dysfunction

Respiratory dysfunction following laparoscopic approach to cholecystectomy is less severe than after open technique, but however a slight impairment of diaphragmatic function occurs[32]. Respiratory

dysfunction is greater in obese, elderly, smokers and patients with COPD.

(d) Postoperative Nausea And Vomiting

Postoperative nausea and vomiting is one of the complications that can delay the discharge of patients after laparoscopic surgeries. Intravenous drainage of gastric contents, administration of droperidol, 5-hydroxytryptamine and liberal intravenous fluid therapy can decrease the symptoms of nausea and vomiting.

Complications Of Laparoscopic Cholecystectomy

The overall mortality rate is 0.1 to 1 per 1000 cases. Bowel perforation, common bile duct injury and significant haemorrhage are some of the major complications. Large vessel injury causing retroperitoneal hematoma can result in significant bleeding without intraperitoneal effusion. Anaesthesiologist must be aware of the occurrence of these complications, and their timely management is vital.

DISTRIBUTION AND PHYSIOLOGICAL RESPONSES MEDIATED THROUGH α_2 ADRENOCEPTORS

Adrenoceptors are membrane bound receptors that mediate the responses of catecholamines, adrenaline and noradrenaline[33]. In 1948, Ahlquist classified them into two distinct classes: alpha (α) and beta (β) [34]. Alpha adrenoceptors were further divided into α_1 and α_2 based on

their anatomical location .The post synaptic α adrenoceptor which mediated responses in the effector organ was designated as α_1 and the presynaptic α adrenoceptor which regulated the release of noradrenaline was designated as α_2[35]. α_2 adrenoceptors are widely distributed throughout the CNS and peripheral tissues and they control the modulation of sympathetic nervous system . α_2 adrenoceptors include three highly homologous subtypes α_{2A}, α_{2B} and α_{2C} , which are required for normal regulation of presynaptic neurotransmitter release from sympathetic nerves in the heart and from the central noradrenergic neurons[36].

α_2 adrenoceptors are G-protein coupled receptors that modulate cellular activity through a second messenger signalling or ion channel modulation activity .Activation of α_2 adrenoceptors coupled to G_i and G_o proteins results in the inhibition of adenylyl cyclise and subsequent decrease in 3' 5' –cyclic adenosine monophosphate (cAMP) formation. This reduces protein kinase activity resulting in reduced phosphorylation of regulatory proteins. Stimulation of phospholipase A2 activity , Na^+/ K^+ exchange and arachidonic acid metabolism are other second messenger systems associated .At high concentrations of α_2 agonists , α_2 adrenoceptors couple with stimulatory Gs proteins resulting in increased adenylyl cyclase activity and increased intracellular Ca^{2+} , which could probably be the reason for smooth muscle contracting effects of α_2 agonists[37]. Activation of G-protein gated K^+ channels

produces cell membrane hyperpolarisation, leading to a reduction firing rate of excitable cells and inhibition of neurotransmitter release. G-protein coupled α_2 adrenoceptor activation also leads to a decrease in Ca^{2+} conductance through direct regulation of voltage gated Ca^{2+} ion channels that results in decreased neurotransmitter release from the nerve terminal[38].

In the CNS, the α_{2A} and α_{2C} receptor subtypes predominate, whereas in the periphery the predominant subtypes are α_{2A} and α_{2B}. The locus ceruleus of the brainstem, an area important in regulating vigilance, attention and stress response, has high density of α_2 receptors; which contributes to the hypnotic and sedative effects of α_2 agonists. The dorsal motor nucleus of vagus nerve also has a high density of α_2 adrenoceptors and this may have a crucial role in the centrally mediated cardiovascular effects associated with α_2 adrenoceptor activation. High densities of α_2 adrenoceptors in areas such as the nucleus tractus solitarius, parts of the ventrolateral medulla and the raphe pallidus may also contribute to cardiovascular changes. The substantia gelatinosa and the intermediolateral cell column in the spinal cord also have α_2 agonist binding sites, responsible for the analgesic effects. The α_2 adrenoceptor in the vascular beds are responsible for vasoconstriction[39].

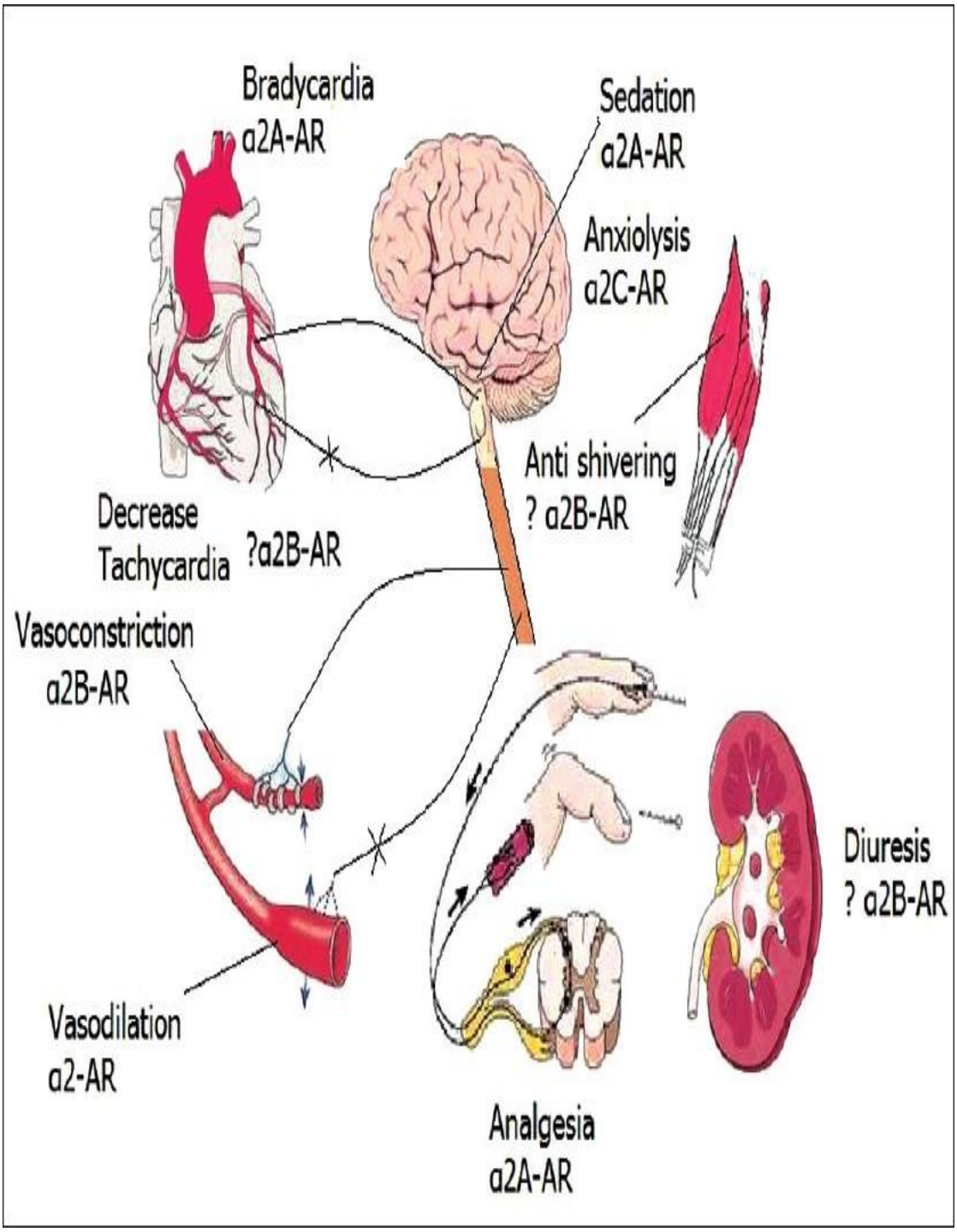


Figure 4: Location and physiological responses mediated through α_2 adrenergic receptors

LOCATIONS AND PHYSIOLOGIC RESPONSES MEDIATED THROUGH A2 ADRENERGIC RECEPTORS[40]

Location of α2 Receptors	Response mediated through α2 Receptors
Central nervous system	Inhibition of neurotransmitter release leading to decreased neuronal firing causing bradycardia, hypotension,sedation,sleep and analgesia
Vascular	Smooth muscle contraction by direct action leading to vasoconstriction Vasodilatation due to central sympatholysis Platelet aggregation
Gastrointestinal tract	Decreases salivation, intestinal secretion and bowel motility
Pancreas	Decreases insulin release
Hypothalamus	Increases growth hormone release
Adipose tissue	Inhibits lipolysis
Kidney	Inhibits renin release Increases glomerular filtration Increases secretion of Na+ and H20
Eye	Decreases intraocular pressure

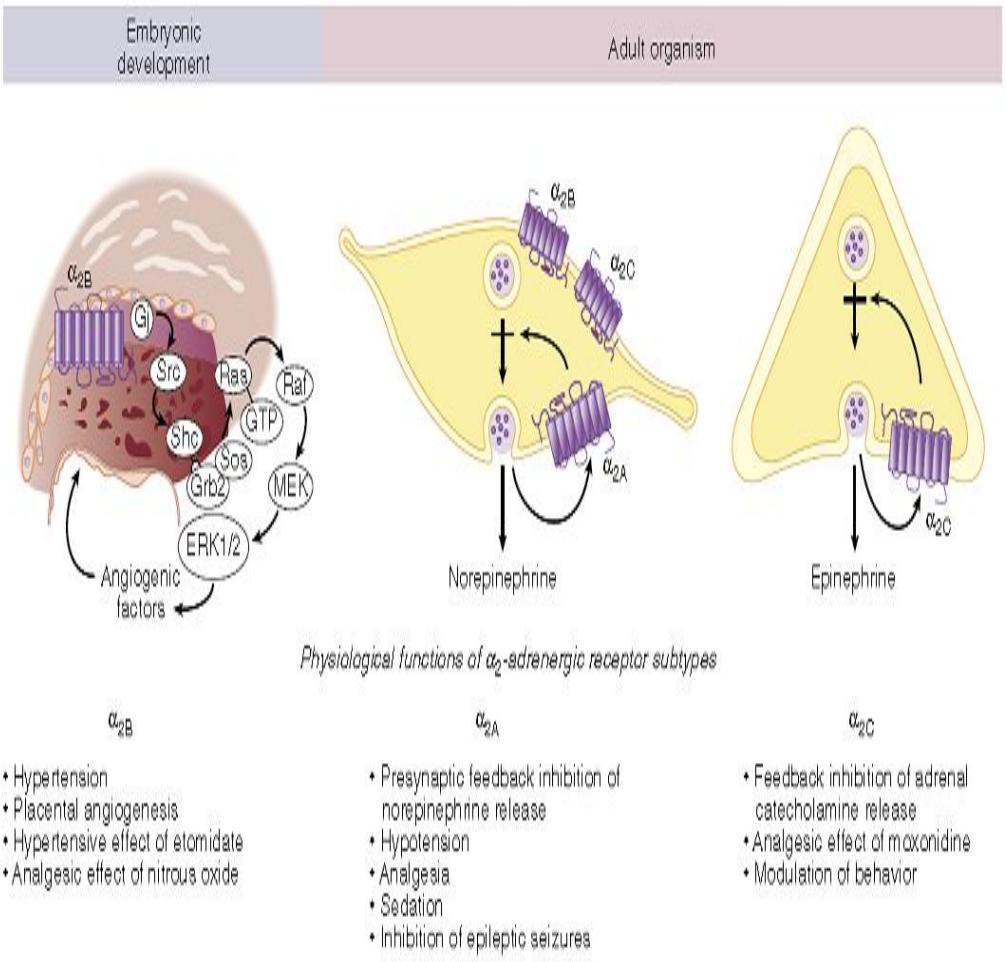


Figure 5: The different physiologic functions of α2 adrenoreceptors

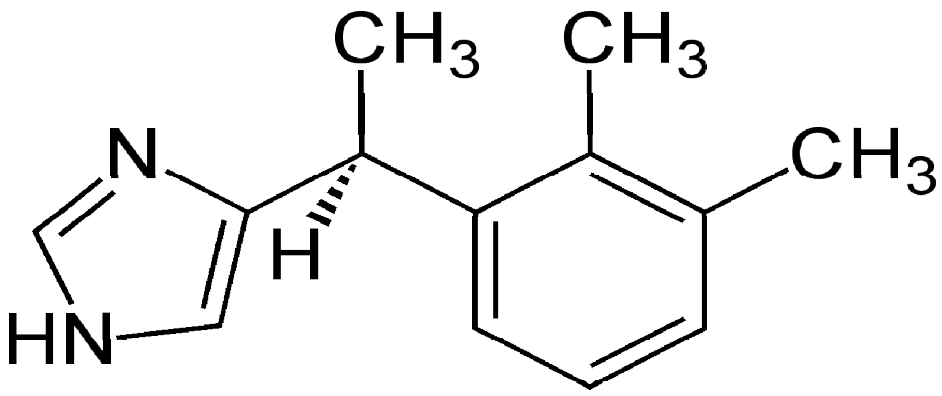
The *top panel* depicts the three α2 receptor subtypes acting as presynaptic inhibitory feedback receptors to control the release of norepinephrine and epinephrine from peripheral or central adult neurons. Also, a negative feedback loop has been seen in the adrenal gland. Alpha2B receptors have been involved in the development of the placental vascular system during prenatal development. The *lower panel* lists a series of physiologic effects with its associated α2 adrenoreceptors.

DEXMEDETOMIDINE

Dexmedetomidine is an α_2 adrenergic agonist which produces sedation, anxiolysis, hypnosis, analgesia, and sympatholysis. Dexmedetomidine is a highly selective α_2 agonist with 1600 times greater selectivity for the α_2 receptor compared with the α_1 receptor.

Physicochemical Characteristics

Dexmedetomidine is a d-enantiomer of medetomidine, belonging to the imidazole subclass of α_2 receptor agonists. It is highly water soluble. It has a high specificity for α_2 receptor (α_2/α_1 1600:1), when compared to clonidine (α_2/α_1 200:1).



Pharmacokinetics

Dexmedetomidine, the dextroisomer of medetomidine, is short acting with linear concentration dependent kinetics.

(i) DOSING AND ADMINISTRATION

Desirable pharmacodynamic effects are observed at a plasma concentration of 0.5-1.2 ng/ml. The dosing regimen approved by FDA in 1999 is a loading dose of 1 µg/kg administered over a period of 10 minutes followed by continuous intravenous infusion at a rate of 0.2-0.7µg/kg/hr. The rate of intravenous infusion should be titrated according to the desired level of sedation. The FDA approved duration of infusion is 24 hours for ICU sedation. Dexmedetomidine when administered for less invasive procedures such as ophthalmic surgeries, is given at a loading dose of 0.5 µg/kg over 10 minutes followed by a maintenance infusion which is started at 0.6 µg/kg/hr and titrated to achieve desired clinical[41].

(ii) DISTRIBUTION

The pharmacokinetics of Dexmedetomidine is commonly described using a two-compartment model .It is rapidly distributed after administration with a distribution half life of 6 minutes. The terminal elimination half life is approximately 2 – 2.5 hours. The steady state volume of distribution is approximately 68 L to 72 L. Dexmedetomidine is highly bound to plasma proteins (94%) without significant variations in pharmacokinetic parameters between males and females[42].

(iii) METABOLISM AND ELIMINATION

Dexmedetomidine is extensively metabolised in the liver through glucuronide conjugation and biotransformation by the cytochrome P450 system without formation of toxic metabolites. The resulting methyl and glucuronide conjugates are excreted by the kidneys. Dexmedetomidine is metabolised by various metabolic pathways. Direct N-glucuronidation to inactive metabolites accounts for 41% of metabolism of Dexmedetomidine. N-methylation to produce 3-hydroxy N-methyl-Dexmedetomidine is the next major pathway accounting for 21% of metabolism of Dexmedetomidine. Hydroxylation followed by conjugation is the other metabolic pathway of Dexmedetomidine. It undergoes conjugation (41%), n-methylation (21%), or hydroxylation followed by conjugation[42, 43].

The terminal elimination half life of Dexmedetomidine is 2 hours. The average clearance value for Dexmedetomidine is approximately 45 L/hr in adults. Renal and hepatic diseases greatly impair the pharmacokinetic properties of Dexmedetomidine. Hepatic impairment results in an increase in the volume of distribution and half-life of Dexmedetomidine as well as a decrease in clearance and protein binding. Renal dysfunction leads to a decrease in the elimination half-life, however the volume of distribution and clearance are not affected[44].

Molecular Weight	236.7 Daltons
Lipid solubility	30
Distribution t1/2	6 min
Protein Binding	94%
Volume of distribution	118 L
Elimination t1/2	120-180 min
Context sensitive half time	4 – 250 min

Pharmacodynamics

(i) MECHANISM OF ACTION

The mechanism of action is unique and different from currently administered sedative agents. Dexmedetomidine acts by activation of α_2 adrenoceptors located in the presynaptic terminal and inhibits the release of norepinephrine. Activation of α_2 adrenoceptors located in the post synaptic terminal in the CNS inhibits sympathetic activity and can cause sedation, anxiolysis along with reduced blood pressure and heart rate. α_2 receptors inhibit adenylyl cyclase activity and result in decreased intracellular cyclic adenosine monophosphate(cAMP) levels. This inhibition of adenylyl cyclise activity is transduced by the inhibitory regulatory protein G_i.

(ii) EFFECTS ON THE CENTRAL NERVOUS SYSTEM

The pivotal neuroanatomic locus for the sedative – hypnotic actions of α2 agonists is the locus ceruleus (LC) of the brain. Hyperpolarisation of noradrenergic LC neurons also occurs in Non-Rapid Eye Movement (NREM) sleep, which involves distinct neuronal pathways .Thus Dexmedetomidine appears to act by activation of NREM sleep .Dexmedetomidine exerts a sedative – hypnotic effect by its action on α_2 receptors in the locus ceruleus and analgesic action by action on α_2 receptors within the locus ceruleus and spinal cord.

The quality of sedation produced by Dexmedetomidine is unique in that, patients are easy to wake up and can follow commands while being tracheally intubated[45]. The α_2 agonists' actions are readily reversed by α_2 adrenergic antagonists (e.g.atipemazole) [46]. It has recently been suggested that α_2 agonists produce sedation through endogenous sleep promoting pathways. Dexmedetomidine reduces the activity of projections of the locus ceruleus (LC) to the ventrolateral preoptic nucleus (VLPO); resulting in an increase in the GABAergic and galanin release in the tuberomammillary nucleus, producing a decrease in histamine release in cortical and subcortical projections. Dexmedetomidine also inhibits ion conductance through L-type or P-type calcium channels and facilitates conductance through voltage gated calcium-activated potassium channels. Dexmedetomidine maintains the

cognitive and immunological function in the sleep deprived states, as in the intensive care unit (ICU) as the hypnosis induced resembles NREM sleep.

Dexmedetomidine is also reported for use in opioid detoxification, cocaine withdrawal, benzodiazepine and opioid tolerance after prolonged sedation. Dexmedetomidine in animal models has CNS protective effects through reduced intracerebral catecholamine outflow, modulation of proapoptotic and antiapototic proteins and the reduced release of excitatory neurotransmitter, glutamate during injury[47, 48, 49]. However, CNS protective effects in humans have not been well documented.

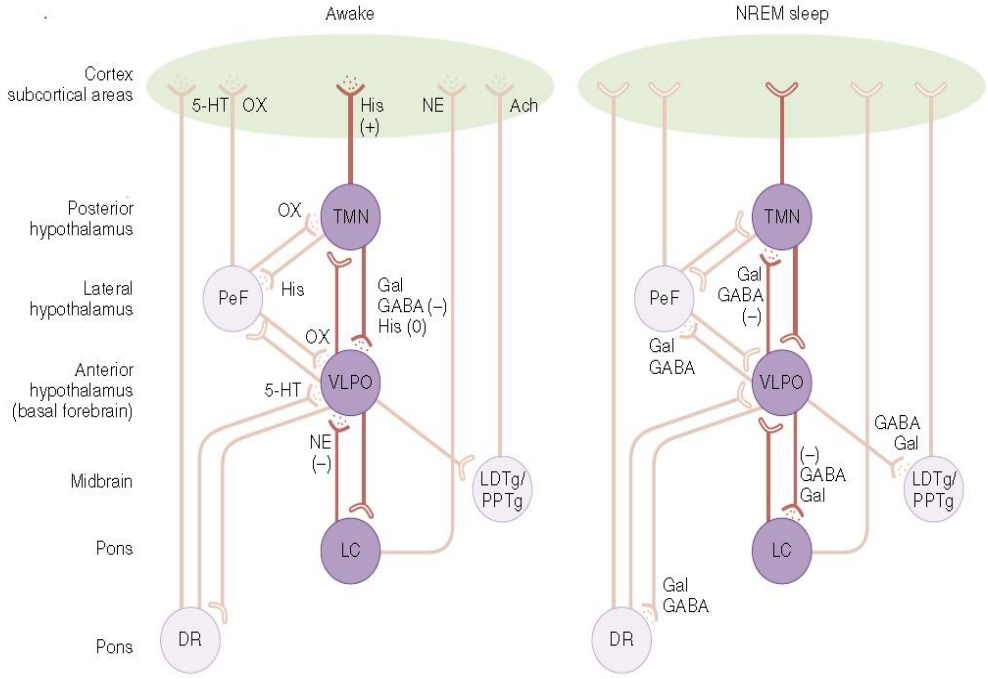


Figure 6: Dexmedetomidine has been shown to induce a non–rapid eye movement sleeping pattern (NREM).

The stimulation of the locus caeruleus (LC) by dexmedetomidine (*right diagram*) releases the inhibition the LC has over the ventrolateral preoptic nucleus (VLPO). The VLPO subsequently releases gamma aminobutyric acid (GABA) onto the tuberomammillary nucleus (TMN). This inhibits the release of the histamine on the cortex and forebrain which prevents arousal and induces the loss of consciousness.

(iii) ANALGESIA

Dexmedetomidine has complex analgesic effects. There are two predominant mechanisms to achieve analgesia namely, activation of descending spinal inhibition and direct activation of presynaptic α_2 receptors on sensory afferent terminals in the dorsal horn. One of the major descending analgesic systems in the CNS utilizes a noradrenergic system that originates in the brainstem locus ceruleus (LC) and terminates on the dorsal horn of spinal cord.

The binding of α_2 agonists in the LC results in norepinephrine release from its descending inhibitory track onto presynaptic terminals of C and Aδ type primary afferent neurons. The subsequent activation of presynaptic α_2 rectors by norepinephrine in the dorsal horn reduces evoked release of excitatory amino acids and neuropeptides from nociceptive neurons, resulting in an analgesic effect. Another property possessed by systemic administration of Dexmedetomidine is its narcotic sparing effects.

Analgesic effects are principally mediated through α_2 receptors when the drug is injected via intrathecal or epidural route[50]. The analgesic effects of α_2 adrenergic agonists and opioids are mediated by different routes, and hence produce a synergistic analgesic action without increasing the incidence of respiratory depression. This, opioid sparing effect is associated with decreased incidence of side effects such as nausea, vomiting and respiratory depression. Systemic α_2 adrenergic agonists may be administered as a perioperative supplement to improve loco regional analgesia. Perioperative Dexmedetomidine also increases the duration of local anaesthetic action in spinal or peripheral nerve block[51, 52]. Some of the systemic analgesic effects of Dexmedetomidine are attributed to the confounding sedative effects. The analgesic-sparing effect observed after a perioperative administration usually lasts up to 24 hours, with the anxiolytic, sedative, and thymoanaleptic properties implicated as being partly responsible for this effect[53, 54].

(iv) EFFECTS ON RESPIRATORY SYSTEM

Sedation producing concentrations of Dexmedetomidine produce a decrease in minute ventilation, but the ventilator responses of CO_2 are retained[55]. Higher concentrations produce a 20 % increase in $PaCO_2$.

(v) EFFECTS ON CARDIOVASCULAR SYSTEM

α_2 agonists are characterized by varied effects on the cardiovascular system including decreased heart rate and systemic vascular resistance and indirectly decreased myocardial contractility, cardiac output, and systemic blood pressure. Bolus dose of Dexmedetomidine results in a biphasic hemodynamic response, producing an initial increase in blood pressure due to direct stimulation of peripheral α_2 receptors and a decrease in heart rate. The heart rate returns to baseline in 15 minutes and the blood pressure reduces to about 15% of baseline values in about 60 minutes. Administration of loading dose leads to increased incidence of hypotension and bradycardia, which can be avoided by omitting the loading dose, or reducing the loading dose to 0.4µg/kg, or administering the loading dose slowly over 20 minutes .Centrally, α_2 agonist administration reduces blood pressure by attenuating sympathetic nervous system tone through a decrease in both spontaneous and evoked sympathetic activity. Stimulation of central postsynaptic α_2 adrenoceptors result in the inhibition of sympathetic outflow and the subsequent decreases in plasma catecholamine concentrations through action at the regulatory centres in the brainstem such as locus ceruleus and nucleus tractus solitarius. Activation of presynaptic α_2 receptors reduces the release of catecholamines from the neuroeffector junction. The decrease in blood pressure corresponds to decrease in plasma catecholamine concentrations. At the same time, α_2

adrenoceptors in the periphery cause vasoconstriction leading to increased systemic vascular resistance .The overall effect on blood pressure is the sum of these two opposing effects.

Dexmedetomidine exhibits no direct depressant effects on the contractile properties of isolated myocardium. In animal models Dexmedetomidine showed some beneficial effects on the ischemic heart through decreased oxygen consumption and redistribution of coronary flow from nonischemic zones to ischemic zones after acute brief occlusion. The perioperative use of α_2 agonists reduces the incidence of perioperative myocardial ischemia. Overall a reduction in myocardial oxygen demand and a decrease in coronary perfusion pressure results as a consequence of hypotension. On balance myocardial energetic are usually improved; however in some patients hypotension may produce myocardial ischemia[56].

Depression of both spontaneous and evoked sympathetic activity in the vasomotor centre of the brainstem occurs at therapeutic doses of α adrenergic agonists, given by oral, intravenous (slowly) or epidural routes of administration .Hence sympatholysis is the predominant hemodynamic effect. Stimulation of α_{2B} adrenoceptors in the peripheral vasculature produces peripheral vasoconstriction. Unlike clonidine, no rebound hypertension has been found with the discontinuation of Dexmedetomidine infusion even when it is given for more than 24

hours. As clinically used α_2 adrenergic agonists possess an imidazoline structure, some of the hemodynamic effects that are observed may be produced by the imidazoline I_1 receptors in the rostral ventrolateral medulla. Hence hypotension induced by α_2 adrenergic agonists cannot be completely reversed by the α_2 adrenergic antagonist Yohimbine, which has a non imidazoline structure but by vasopressors and inotropes. There appears to be an enhanced pressor response to Ephedrine, Phenylephrine and Dobutamine, but not to Norepinephrine[57]. The effects of α_2 adrenergic agonists are readily reversible by the α_2 adrenergic antagonist Atipemazole.

Uses

(i) INTENSIVE CARE UNIT

Dexmedetomidine has been approved for sedation in mechanically ventilated patients and lesser amounts of opioids are required, when Dexmedetomidine is used for sedation compared to propofol or benzodiazepines. It produces unique characteristics of sedation with minimal respiratory depression. Also, Dexmedetomidine when used for sedation provides a more stable hemodynamics during weaning[58].Dexmedetomidine is used for the treatment of withdrawal of narcotics, benzodiazepines, alcohol, and recreational drugs[59].

(ii) ANAESTHESIA

Dexmedetomidine has been used as a premedicant in doses of 0.3 - 0.6 µg/kg , reducing the requirements of intravenous and volatile anaesthetics and also attenuating the hemodynamic stress response to endotracheal intubation [60,61].Dexmedetomidine is also used for sedation in patients for monitored anaesthesia care .When used as a premedication for cataract surgeries , intraocular pressure decreases, catecholamine secretion decreases and also perioperative analgesic requirements are lesser, recovery rates are rapid . For maintenance of anaesthesia, Dexmedetomidine is used in infusion rates of 0.2-0.7 µg/kg/hr. This reduces postoperative pain scores, morphine consumption and improved hemodynamics when compared with desflurane - fentanyl or propofol – fentanyl anaesthetics[62, 63].

This highly selective α_2 agonist has unique characteristics that include titrable sedation, sympatholysis and analgesia without significant respiratory depression, which has led to a dramatic increase in its use in many off – label applications in the ICU, operating room, and perioperative environment.

CHAPTER 3

REVIEW OF LITERATURE

Aantaa R et al[64] (Anaesthesiology 1997) conducted a study to determine the effects of Dexmedetomidine on the minimal alveolar concentration (MAC) of isoflurane. MAC of isoflurane was chosen as the measure of anaesthetic potency in his study. The study was conducted on 49 women posted for abdominal hysterectomy. Patients were randomized into two groups. The patients in the first group received placebo infusion. A two-stage infusion of Dexmedetomidine was used in the second group with a target plasma concentration of 0.3 ng/ml or 0.6 ng/ml. Dexmedetomidine infusion was started 15 minutes before the induction of anaesthesia and was continued until skin closure. Induction of anaesthesia was performed with thiopental and alfentanil. Up-down method of Dixon was used to predetermine the end tidal concentration of isoflurane. This end-tidal concentration was maintained for 15 minutes and then the patient's response to skin incision was assessed. In the control group the MAC of isoflurane was 0.85% end-tidal. In the Dexmedetomidine 0.3 ng/ml group the MAC was 0.55% end-tidal and in the Dexmedetomidine 0.6 ng/ml group the MAC was 0.45% end-tidal. His study concluded that MAC of isoflurane was significantly lesser in the Dexmedetomidine group when compared to

the control group and also showed that increasing plasma concentrations of Dexmedetomidine resulted in progressive decreases in MAC of isoflurane.

Ebert Thomas et al[65] (Anaesthesiology 2000) conducted a study to evaluate the effects of increasing plasma concentrations of Dexmedetomidine. The study was conducted on 10 healthy volunteers. The following monitors were used- ECG, pulse oximetry, $EtCO_{2,}$ invasive blood pressure, central venous pressure, pulmonary artery pressure, cardiac output, respiratory rate, arterial blood gases and plasma catecholamine concentrations. During the study specific tests were performed at every 40 minute intervals during the sequential increase in Dexmedetomidine plasma concentrations (0.5, 0.8, 1.2, 2.0, 3.2, 5.0, and 8.0 ng/ml; baroreflex testing only at 0.5 and 0.8 ng/ml). Hemodynamic parameters and blood gases were observed. Psychometric tests, cold pressor and baroreflex tests were performed. He observed the following results. Plasma catecholamine concentrations were decreased at all doses of Dexmedetomidine. Dexmedetomidine in plasma concentrations of 0.5 ng/ml and 0.8 ng/ml increased sedation by 38% & 45% respectively and reduced mean arterial pressure by 13%. Central venous pressure and pulmonary arterial pressure remained unchanged. Progressive increase in plasma concentrations of Dexmedetomidine resulted in progressively increasing sedation, progressive increase in all pressures and systemic vascular resistance

and progressive decrease in heart rate, cardiac output and stroke volume. Progressive increase in plasma concentrations of Dexmedetomidine also resulted in progressively decreasing pain rating scores. Increase in MAP to cold pressor test progressively diminished as the plasma concentrations of Dexmedetomidine diminished. This study concluded that increasing concentration of Dexmedetomidine results in progressively increasing analgesia and sedation, and progressive decreases in heart rate, cardiac output, memory and attenuation of cold pressor response.

Aho et al[66] (Anesthesiology 1991) conducted a study to evaluate the effects of Dexmedetomidine on maintaining hemodynamic stability during the perioperative period and to analyze its effects on isoflurane requirements in patients undergoing abdominal hysterectomy. The study was conducted on 96 women undergoing abdominal hysterectomy, randomized into four groups receiving Dexmedetomidine 0.3 µg/kg, Dexmedetomidine 0.6 µg/kg, fentanyl 2.0 µg/kg, or saline respectively. All the above drugs are administered as a single bolus dose 10 minutes prior to the induction of anaesthesia. Induction was performed with thiopental 4.0 mg/kg. Maintenance of anaesthesia was initiated with Isoflurane 0.3% end-tidal concentration in 70% nitrous oxide. Blood pressures and heart rate were maintained within 20% of baseline values by varying the end-tidal isoflurane concentration. Fentanyl was supplemented if the end-tidal isoflurane concentration exceeded 1.5%.

Increase in blood pressure and heart rate was recorded in all the four groups on tracheal intubation. But the degree of increase was lesser in Dexmedetomidine 0.6 µg/kg group when compared to the saline group ($P<0.01$). Heart rate response to endotracheal intubation was significantly lesser in Dexmedetomidine group when compared to the saline or fentanyl group. The end-tidal isoflurane concentration required was also significantly less in the Dexmedetomidine group (0.35%) when compared to the saline (0.47%) or fentanyl (0.48%) groups. This study concluded that Dexmedetomidine when administered as a single bolus dose prior to induction resulted in significant attenuation of the stress response to endotracheal intubation and also greatly diminished isoflurane requirements when compared to fentanyl.

Feld et al[67] (J Clin Anesth 2006) conducted a study to determine whether Dexmedetomidine could serve as a better alternative to fentanyl for analgesia in patients undergoing bariatric surgery . The study was conducted on 20 patients with an average BMI of 54 to 61 kg/m^2 undergoing gastric bypass surgery. The patients were randomly allocated into two groups. First group received fentanyl bolus of 0.5 µg/kg followed by an infusion at a rate of 0.5 µg/kg/hr. The second group received Dexmedetomidine at a bolus dose of 0.5 µg/kg followed by infusion at a rate of 0.4 µg/kg/hr. In the study bispectral index was maintained at 45-50 by varying end-tidal desflurane concentration. Heart rate and blood pressure were recorded every 5 minutes

intraoperatively. End-tidal desflurane concentration and bispectral index were observed every 60 minutes. Post-operative pain scores and morphine consumption by patient controlled analgesia devices were also determined. Mean blood pressure and heart rate and the average end-tidal desflurane concentration were significantly reduced in Dexmedetomidine group when compared to the fentanyl group. Post-operative pain scores and morphine consumption were also decreased in the Dexmedetomidine group. The study concluded that Dexmedetomidine attenuated blood pressure and provides post-operative analgesia, when used as a substitute to fentanyl during gastric bypass surgery.

Hall et al[68] (Anesth Analg 2000) conducted a study to evaluate the efficacy of two small infusion doses of Dexmedetomidine. They studied the effects of Dexmedetomidine on sedation, analgesia, cognition and respiratory function. The study was conducted on seven healthy volunteers. Informed consent was obtained. The study was conducted in three phases. The participants were randomly assigned to receive two different doses of drug or placebo. The following monitors were used- heart rate, NIBP, respiratory rate, $EtCO_2$, SpO_2 and EEG (bispectral analysis). Baseline hemodynamic parameters were recorded. Various psychometric tests were performed including visual analog scale for sedation, alertness scale, memory and digit symbol substitution test. Cold-pressor test was used to quantify pain. All patients received a

10 minute infusion dose of saline or Dexmedetomidine at a dose of 0.2 or 0.6 µg/kg/hr. All patients were again evaluated at the end of drug infusion and also during the recovery period. Dexmedetomidine infusion resulted in significant sedation and impairment of memory and psychomotor performance with preservation of heart rate, blood pressure, SpO2, EtCO2 and respiratory rate. Pain response to cold pressor test was also suppressed. The study thus concluded that small intravenous doses of Dexmedetomidine provided reversible sedation, analgesia, memory and cognitive impairment without any cardiorespiratory compromise.

Aho et al[69] (Anesth Analg 1992)conducted a study to determine the efficacy of Dexmedetomidine when used as an intravenous infusion for the maintenance of anaesthesia. Patients were anesthetised with thiopental, fentanyl and isoflurane. It was decided to conduct the study in two phases. The first phase was conducted on 14 women undergoing abdominal hysterectomy and it was a dose response study. In this phase the dosing of Dexmedetomidine was determined from the measured hemodynamic parameters. The second phase of the study was then conducted on 20 patients randomized into two groups. One group received placebo and the other group received Dexmedetomidine infusion. Dexmedetomidine was administered as a two staged infusion. The initial dose was administered over 10 minutes prior to the induction of anaesthesia. The maintenance rate was commenced from the time of

induction and was continued till the closure of abdominal fascia. Dexmedetomidine dose ranges in the dose response phase varied from 120 ng/kg/min to 270 ng/kg/min. They chose an initial infusion dose of 170 ng/kg/min for the second phase of the study followed by 10 ng/kg/min for maintenance. During surgery isoflurane was titrated according to predetermined hemodynamic criteria. Dexmedetomidine infusion decreased the requirement of isoflurane by >90%. The endocrine stress response to endotracheal intubation was also blunted. Their study thus concluded that Dexmedetomidine decreased the requirement of isoflurane and also blunted the stress response to endotracheal intubation.

Talke et al[70] (Anesth Analg 1997) designed a study to determine the postoperative pharmacokinetic and sympatholytic effects of Dexmedetomidine. The study was conducted on 8 women. Dexmedetomidine was infused for 60 minutes postoperatively by computer-controlled infusion protocol (CCIP) with a target plasma concentration of 600 pg/ml. Blood sampling was done thrice – before the start of infusion, during and after the infusion and plasma levels of norepinephrine, epinephrine and Dexmedetomidine were also determined. Norepinephrine levels decreased from 2.1 +/- 0.8 to 0.7 +/- 0.3 nmol/L; epinephrine decreased from 0.7 +/- 0.5 to 0.2 +/- 0.2 nmol/L. There was also a significant decrease in the heart rate and systolic blood pressure. The study thus concluded that

Dexmedetomidine attenuates sympathetic activity during the intraoperative and immediate postoperative period.

Arain SR et al[71] (Anesth Analg 2002) designed a comparative study to evaluate the efficacy, adverse effects and recovery characteristics of Dexmedetomidine and propofol. Cardiorespiratory effects were also evaluated. Sedation, postoperative analgesia and psychomotor performance were chosen as secondary end points. The study was conducted on 40 patients randomized into 2 groups. The first group received Dexmedetomidine at a loading dose of 1 µg/kg administered over 10 minutes followed by an infusion dose of 0.4-0.7 µg/kg/min. The second group received propofol – loading dose of 75 µg/kg/min over 10 minutes followed by a maintenance dose of 12.5-75 µg/kg/min. Heart rate and mean blood pressure were studied every 5 minutes. Sedation was assessed with visual analogue scale and Observer Assessment of Alertness scale. Respiratory rate, SpO_2, $EtCO_2$ were also monitored. Psychomotor performance was analyzed using digital symbol substitution test. Pain was assessed using visual analogue scale. Intraoperative sedation levels were targeted to maintain a bispectral index score of 70-80. Sedation was achieved more rapidly with propofol when compared to Dexmedetomidine.The average infusion rate was 0.7 µg/kg/hr for Dexmedetomidine and 38 µg/kg/min for propofol. Psychomotor performance and respiratory rate showed no difference between two groups. The study concluded that Dexmedetomidine causes

more sedation, results in lower blood pressure and improved analgesia when compared to propofol.

Alp Gurbet et al[72] (Can J ANESTH 2006) designed a study to evaluate the efficacy of Dexmedetomidine in providing post operative analgesia. Post operative pain scores and morphine consumption were studied. The study was conducted on 50 women posted for total abdominal hysterectomy. The patients were randomly assigned into two groups. The first group received Dexmedetomidine at a loading dose of 1 µg/kg followed by maintenance at a rate of 0.5 µg/kg/hr. The second group received placebo infusion. Heart rate SpO_2, systolic and diastolic blood pressures were monitored. Morphine was administered post operatively by patient controlled analgesia device. Pain and sedation scores and total morphine consumption were studied for 48 hours following surgery. Pain and sedation scores between the two groups were similar. Patients in the Dexmedetomidine group required significantly less morphine when compared to the placebo group. The study thus concludes that Dexmedetomidine provides effective post operative analgesia, and reduces morphine consumption post operatively without increasing the incidence of adverse effects.

Talke et al[73] (Anaesthesiology 1995) studied the perioperative effects of Dexmedetomidine in patients undergoing vascular surgery. The study was conducted on 24 patients undergoing vascular surgery.

Patients were randomized into four groups. The first group received placebo. The second group received low dose Dexmedetomidine (0.15 ng/ml). The third group received medium dose Dexmedetomidine (0.30 ng/ml). The fourth group received high dose Dexmedetomidine (0.45 ng/ml). The infusion was started 60 minutes prior to induction and was continued till 48 hours post operatively. The patients were monitored with NIBP, heart rate, SpO_2, ECG, anaesthetic concentrations, intraoperative ECHO. Post operative cardiac enzymes were also studied. There was a progressive decrease in heart rate and systolic blood pressure as the dose of Dexmedetomidine progressively increased. Post operatively Dexmedetomidine groups had less tachycardia when compared to the placebo group. Also intraoperatively patients in the Dexmedetomidine groups required more vasoactive interventions to maintain the hemodynamic parameters within the desired range. This study showed that Dexmedetomidine upto a plasma concentration of 0.45 ng/ml has a beneficial effect on perioperative hemodynamic management, but requires more pharmacological interventions to maintain blood pressure and heart rate within the desired limits.

Tufanogullari et al[74] (Anaesthesia –Analgesia, 2008) conducted a study to evaluate the effects of Dexmedetomidine on both early and late recovery. Eighty morbidly obese patients posted for laparoscopic bariatric surgery were chosen. Informed consent was obtained. The patients were randomly assigned to 4 groups. The first group received

saline infusion. Dexmedetomidine 0.2 group received an infusion of 0.2 µg/kg/hr IV. Dexmedetomidine 0.4 group received an infusion of 0.4 µg/kg/hr IV and Dexmedetomidine 0.8 group received an infusion of 0.8 µg/kg/hr. Desflurane concentrations were titrated to maintain mean arterial pressure values within 25 % of baseline values. Hemodynamic parameters, postoperative pain scores, and the need for "rescue" analgesics and antiemetics were studied. Average end-tidal desflurane concentrations progressively decreased by 19%, 20% and 22% as the concentration of Dexmedetomidine is progressively increased respectively. Blood pressures and the length of stay in PACU were significantly reduced in Dexmedetomidine groups when compared to the saline group. The need for rescue fentanyl and antiemetic therapy was also reduced in Dexmedetomidine group when compared to the saline groups. The study thus concluded that adjunctive administration of Dexmedetomidine in doses of 0.2-0.8µg/kg/hr decreased fentanyl use, antiemetic therapy, and the length of stay in the PACU.

Hirvonen EA et al[75] (Surgical endoscopy 2000) conducted a study to determine the effects of patient positioning, anaesthesia and increased intraabdominal pressure during laparoscopy on cardiac performance. 15 ASA class I patients posted for laparoscopic cholecystectomy were chosen. Using invasive monitoring, hemodynamic changes were investigated. Cardiac index (CI), stroke index (SI), central venous pressure (CVP), pulmonary capillary wedge

pressure (PCWP), and systemic vascular resistance were monitored. Measurements were first taken in the supine position and then in the head-up position (15-20 degrees) both when awake and after the induction of anaesthesia. Head-up tilt resulted in decreased cardiac index (CI), stroke index (SI), central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and increased systemic vascular resistance. Carbon dioxide insufflation resulted in increases in CVP and PCWP, systemic and pulmonary arterial pressures. CI or SI remains unchanged. Deflation of gas results in normalization of these hemodynamic parameters. The study thus concluded that head – up positioning accounts for many of the adverse effects in hemodynamics during laparoscopic cholecystectomy.

Jorris J et al[76] (Anesth Analg 1993) conducted a study to determine the hemodynamics (invasive arterial pressure , PAP, RAP , PCWP , HR ,CO , SVR and pulmonary vascular resistance) during laparoscopic cholecystectomy.15 ASA class I patients were chosen. Intra-abdominal pressure was maintained constant at 14 mmHg. Hemodynamics measurements were made preoperatively, after the induction of anaesthesia, head up tilt, 5, 15 and 30 minutes after peritoneal insufflation, and 30 minutes after exsufflation . Head-up tilt resulted in a significant reduction in cardiac preload and caused further reduction of CI. Peritoneal insufflations caused a significant increase of mean arterial pressures (35%), a significant reduction of CI (20%), and a

significant increase of systemic (65%) and pulmonary (90%) vascular resistances. The combined effect of anaesthesia, head-up tilt, peritoneal insufflations produced a 50% decrease in CI. This study concludes that laparoscopic cholecystectomy in the head-up position results in significant hemodynamic changes, mainly at the beginning of peritoneal insufflation.

CHAPTER 4

MATERIALS AND METHODS

This study on patients undergoing laparoscopic cholecystectomy was approved by the Institutional Ethical Committee, Stanley Medical College, Chennai. This was a prospective study conducted on 60 patients over a period of 1 year. Pre-anaesthetic evaluation was done, recording a detailed history and performing a complete physical examination. Complete blood count, renal function test, blood grouping/typing, random blood sugar, HBsAg, HCV and anti retroviral screening tests were done. After discussion of anaesthetic options, a written preoperative consent was obtained.

SAMPLE SIZE AND RANDOMIZATION

The sample size was calculated as 60 based on the pilot study and statistical reports from previous studies. The group sizes (n =30) were calculated to detect a reduction in the total fentanyl requirement and /or average inspiratory desflurane concentrations, with a power of 90% [assuming a variability (sd) of ±10%] and a significance level of 0.05. They were randomly allocated to 30 in each group and were named as Group A (Dexmedetomidine 0.2 µg/kg/hr) and Group B (0.6 µg/kg/hr). The investigator prepared 60 lots numbered serially from 1-60. A coding sheet was also simultaneously prepared that allotted each number

randomly to a group. The observer is allowed to take a lot and the selected number was marked in the proforma .Then the observer is blinded for dose of drug being infused and performs the procedure. At the end of the study coding sheet was revealed. For the serial numbers which were selected and excluded as per the exclusion criteria, the same serial number is mixed again in the lot by the investigator.

(i) INCLUSION CRITERIA

(a) ASA grade I

(b) Age 18 to 55 years

(c) Weight 60 ± 20 kg

(d) Both Sex

(e) Undergoing Endotracheal General Anaesthesia

(f) Laparoscopic Cholecystectomy

(g) Without any co morbid illness

(ii) EXCLUSION CRITERIA

(a) Allergy to alpha -2 adrenergic agonist or sulpha drugs

(b) Clinically significant cardiovascular ,neurologic ,renal, hepatic or gastrointestinal diseases

(c) Pregnant and nursing women

(d) Morbid obesity

(e) Hypertensive patients on beta blockers

(f) Diabetes

(g) Heart block

(h) History of alcohol or drug abuse

CONDUCT OF ANAESTHESIA

Groups

Group A : Patients receiving desflurane – fentanyl – Dexmedetomidine infusion at 0.2 µg/kg/hr

Group B : Patients receiving desflurane – fentanyl – Dexmedetomidine infusion at 0.6 µg/kg/hr

Monitoring

Monitors that were made available included

(a) NIBP

(b) SPO_2

(c) ECG

(d) $ETCO_2$

(e) HR

Methodology

60 patients within the age of 18 – 55 years undergoing laparoscopic cholecystectomy under endotracheal general anaesthesia were randomized into two groups of 30 each.

All patients were fasted for 8 hours before the planned surgical procedure. Tab. Ranitidine 150mg, Tab. Metoclopramide 10mg and Tab. Diazepam 5mg were given orally on the night before surgery and at 6 am on the day of surgery with sips of water. On the day of surgery all patients were shifted to the premedication room and a peripheral intravenous line was secured using 18 G IV cannula.

Preparation of Study Medication

Infusion of the study medication was prepared (100 µg of Dexmedetomidine added to saline to achieve a total volume of 50ml – 2 µg / ml preparation). The weight adjusted doses were based on patients' actual body weight.

Premedication

All patients were premedicated with Inj. glycopyrrolate 0.2 mg IM 30 minutes before entering the operating room.

The patients' baseline heart rate, BP, SPO2, RR were recorded 5 min after arrival in the operating room. Patients SpO_2, NIBP, ECG, ETCO2 and heart rate were monitored.

After obtaining the baseline HR and BP, infusion of the study medication was started at weight adjusted doses according to the patients' actual body weight.

Preoxygenation with 100% oxygen for 3 minutes was started 7 minutes after the commencement of infusion medication.

Inj. Fentanyl at a dose of 1µg/kg was administered before induction of anaesthesia.

Induction

Anaesthesia was induced 10 min after the commencement of study drug infusion with propofol 2 mg / kg IV and muscle relaxation was obtained by Inj. Atracurium at a dose of 0.5 mg/kg. Desflurane was administered at an inspiratory concentration of 3%.

Maintanence

Anaesthesia was initially maintained with nitrous oxide (3 l / min), oxygen (1.5 l /min) mixture with desflurane administered at 3 % inspired concentrations. Ventilation was adjusted to maintain $EtCO_2$ between 30 and 40 mmHg. Muscle relaxation was achieved by

administration of Inj. Atracurium in doses of 0.1 mg/kg IV every 20 minutes.

Intra –operative HR, SBP, DBP, MAP, SpO_2, $EtCO_2$ were recorded every 5 min for first 30 min and subsequently every 10 min intervals till the discontinuation of anaesthetic drugs .Hemodynamic variables were also recorded at specific end –points (induction of anaesthesia, 1 min after induction, intubation, 5 min after tracheal intubation, skin incision, 5 min and 10 min after skin incision).

MAP and heart rate values were maintained within 25% of baseline values by administering bolus doses of fentanyl at 0.5 µg / kg (up to a maximum of 2 µg / kg) and by varying inspiratory desflurane concentrations.

Hypotension (defined as MAP < 25 % of baseline values on two consecutive readings within 2 – 3 min), was first treated by decreasing the desflurane concentration and by administering fluid bolus of 200ml. Hypotension not responding to the above two interventions, was treated with Inj. Ephedrine in bolus doses of 6 mg IV. The infusion of study medication was stopped if hypotension persists > 3 min after these interventions. Upon return of MAP to within 25 % of baseline values the infusion of study medication was resumed at 50 % of initial infusion rate and the inspiratory concentration of desflurane was reset at 3%.

In the presence of hypertension (defined as MAP value > 25 % of baseline values on two consecutive readings within 2 – 3 min) and / or tachycardia (defined as HR > 25 % of baseline values > 2 min), bolus doses of fentanyl at 0.5 ug / kg (up to a maximum of 2 ug / kg) were administered at 10 min intervals. Hypertension and tachycardia not responding to fentanyl boluses were treated by increasing the inspiratory concentrations of desflurane in steps of 1% up to a maximum inspiratory concentration of 6%. Hypertension and tachycardia not responding to the above interventions were treated by administration of propofol in bolus doses of 0.5mg/kg IV.

Bradycardia defined as HR < 50 persisting for > 2 min, was treated with Inj. Atropine 0.6 mg IV boluses.

During the surgical procedure patients received IV crystalloid solutions calculated according to the Holliday Segar formula (4-2-1 formula) and IAP was set at a constant value of 12 mm Hg.

Ondansetron 0.1 mg/kg IV was given for the prevention of post operative nausea and vomiting once the laparoscope is withdrawn.

Infusion of Dexmedetomidine and desflurane administration was discontinued upon the start of wound closure and inspired oxygen flow rate increased to 5 l / min.

After wound closure residual neuromuscular block was reversed with Inj .neostigmine 0.04 mg / kg IV and glycopyrrolate 0.01 mg / kg IV, once the patients started breathing spontaneously. When spontaneous respirations were adequate and patients were able to obey simple commands, oropharyngeal suctioning was done and tracheal extubation was performed.

Times from discontinuation of Dexmedetomidine to eye opening, and tracheal extubation were recorded.

The degree of sedation following tracheal extubation was assessed using the **Ramsay sedation scale**. In this system, 1 = agitated and uncomfortable, 2 = cooperative and orientated, 3 = can follow simple directions, 4 = asleep but strong response to stimulation, 5 = asleep and slow response to stimulation, and 6 = asleep and no response to stimulation.

The average inspiratory desflurane concentrations was calculated as the sum of the products of inspiratory concentrations and times divided by the total anaesthesia time.

All patients were shifted to the post anaesthesia care unit at the end of the surgery and were observed for four hours. Hemodynamic variables such as heart rate, SBP, DBP, MAP and SpO_2 were monitored.

Sedation scores were studied at 30 minute intervals for two hours and then every 60 minutes for the subsequent two hours.

Total fentanyl requirement, average inspiratory desflurane concentrations, HR, SBP, DBP, SPO_2, $ETCO_2$, post operative sedation score and intraoperative requirement for adjuvant such as propofol were studied.

Parameters Studied

All the parameters and results from two groups (Group A and Group B) were entered in the predesigned proforma sheet.

i. Total fentanyl requirement throughout the surgical procedure

ii. The average inspiratory desflurane concentrations calculated as the sum of the products of inspiratory concentrations and times divided by the total anaesthesia time.

iii. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), oxygen saturation (SpO_2), $EtCO_2$ every 5 minutes for the first 30 minutes and then subsequently every 10 minutes till the discontinuation of anaesthetic drugs and also at specific end points(induction, 1 minute after induction, intubation, 1 minute after intubation and peritoneal insufflation).

iv. Sedation scores following extubation, 30 minute intervals for the first two hours following extubation and then at 60 minute intervals for the next two hours.

v. Intraoperative need for adjuvant such as propofol.

vi. Intraoperative need for a fluid bolus and ephedrine administration.

CHAPTER 5

OBSERVATION AND RESULTS

After collecting the data, all the variables are examined for outliers and non-normal distributions. The Categorical variables are expressed as Frequency and Percentage. The Quantity variables are expressed as mean and standard deviation. Descriptive statistics are used to evaluate baseline characteristics.

Student's *t*-test was used to analyze the parametric data, and discrete (categorical) variables were analyzed using the $\chi 2$ test, with a $P < 0.05$ considered statistically significant.

The statistical analysis was carried out using statistical software package SPSS 22.0.

Table 1: Comparison of mean of age, weight and mean duration of surgery between Group A and Group B

	GROUP A	GROUP B	p value
Age	34.07 ±10.81	33.50±10.87	0.837
Weight	58.76±6.01	59.20 ± 5.997	0.536
Duration of surgery	96.55±22.08	100.00±14.14	0.137

The mean age of patients in Group A and Group B was 34.07 years and 33.50 years respectively.

The mean weight of patients in Group A and Group B was 58.76kg and 59.20 kg respectively.

The mean duration of surgery in Group A and Group B was 96.55 minutes and 100 minutes respectively.

On analysing the data statistically, the p value was calculated as p=0.837, p=0.536 and p=0.137 for age, weight and duration of surgery respectively. All these values were >0.05, hence the difference was statistically insignificant between the two groups in terms of age, weight and duration of surgery, and the two groups were therefore comparable.

Figure 7: Bar chart comparing Group A and Group B in mean age, weight and duration of surgery

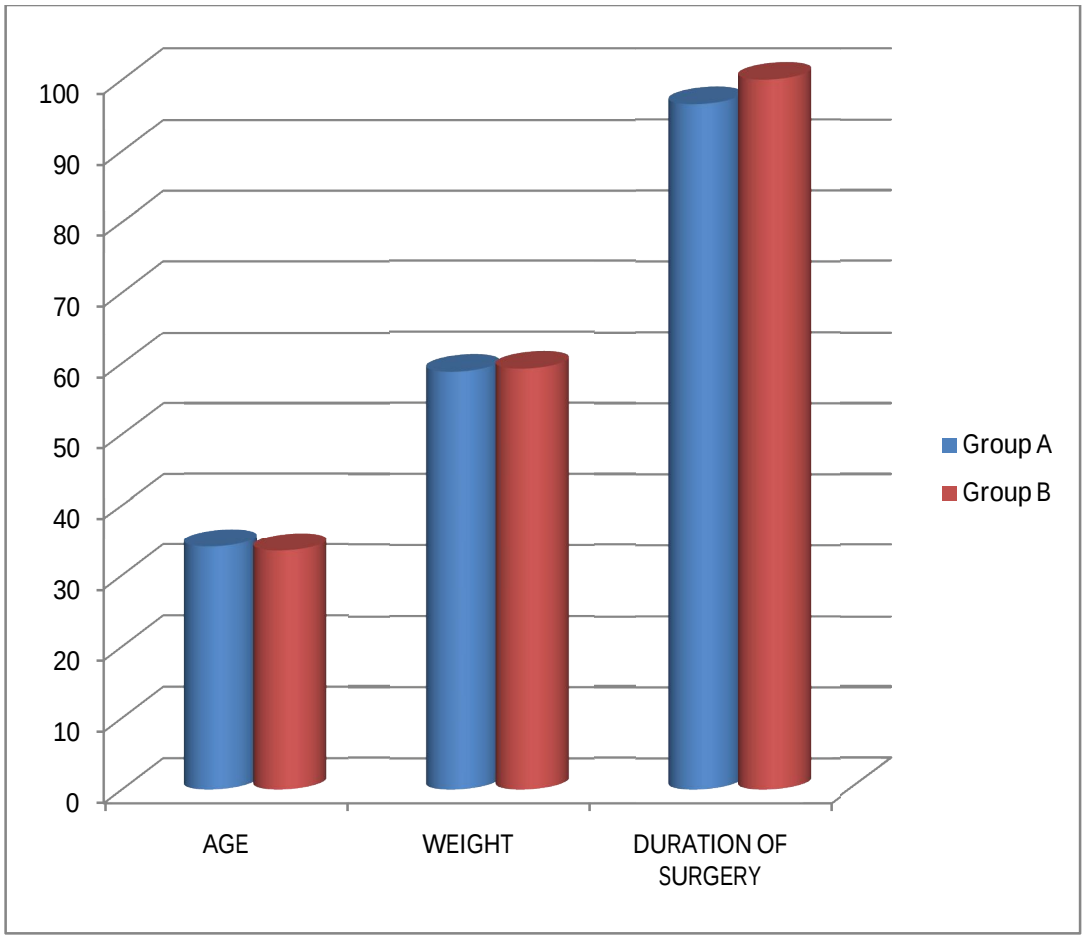


Table 2: Comparison of gender distribution between Group A and Group B

Gender	Group A	Group B	P value
Male	6	7	1.000
Female	24	23	1.000

Total number of males in Group A and Group B was 6 and 7 respectively.

Total number of females in Group A and Group B was 24 and 23 respectively.

On analysing the data statistically, the p value was calculated as p=1.000 for both males and females between the two groups respectively. All these values were >0.05, hence the difference was statistically insignificant between the two groups in terms gender distribution, and the two groups were therefore comparable.

Table 3: Comparison of mean of total fentanyl requirement and average inspiratory desflurane concentrations between Group A and Group B

	Group A	Group B	p value
Total fentanyl	74.31 ± 18.56	60.00 ± 2.99	0.001
Avg. Inspiratory desflurane	2.66 ± 0.091	2.57 ± 0.12	0.002

The mean of total fentanyl required in Group A and Group B was 74.31 µg and 60.00 µg respectively.

The mean average inspiratory desflurane concentration in Group A and Group B was 2.66 and 2.57 respectively.

On analysing the data statistically, the p value was calculated as p=0.001 and p=0.002 for total fentanyl and average inspiratory desflurane concentrations respectively. All these values were <0.05, hence the difference was statistically significant between the two groups in terms of total fentanyl, and average inspiratory desflurane concentrations. Hence we concluded that total fentanyl requirements and the average inspiratory desflurane concentrations were significantly lesser in group B when compared to group A.

Figure 8: Bar diagram comparing the mean of total fentanyl and average inspiratory desflurane concentrations between Group A and Group B

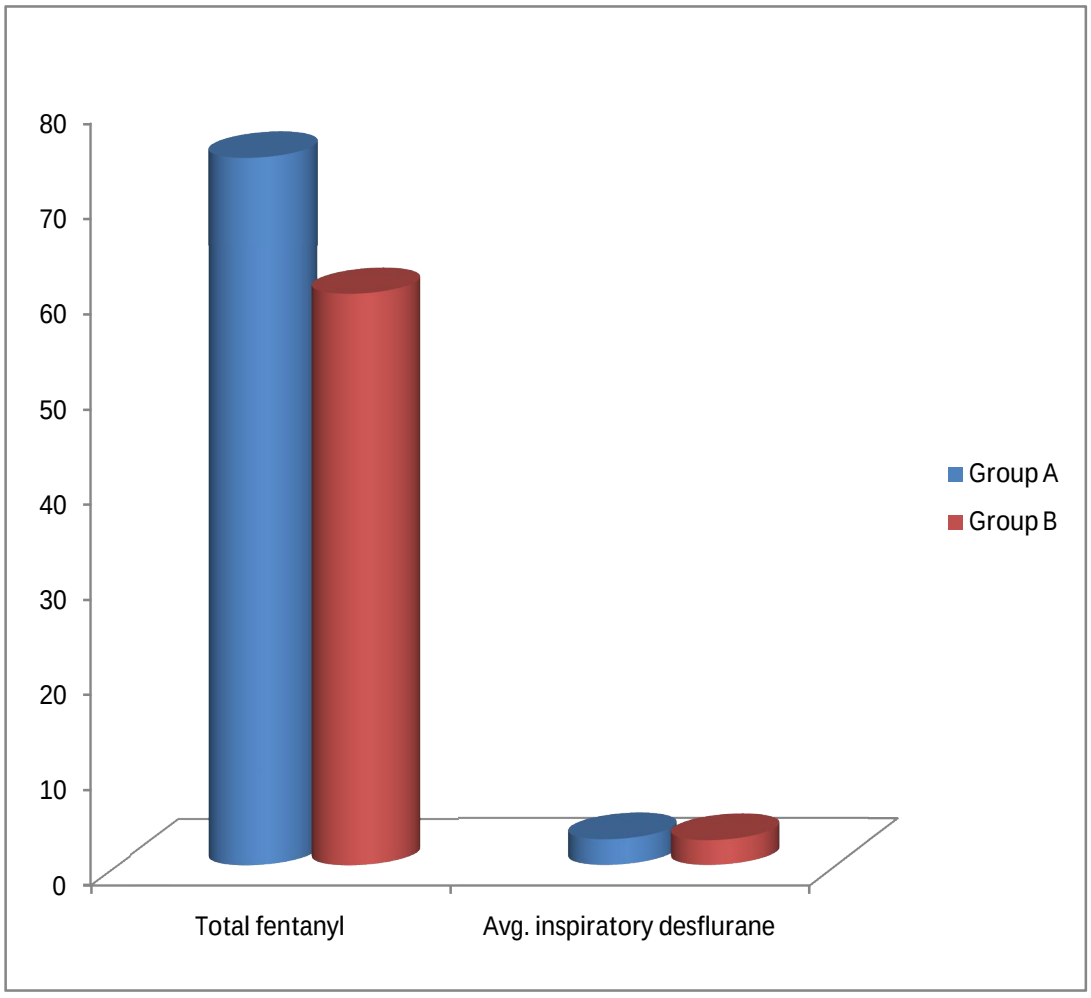


Table 4: Comparison of total number of bolus doses of fentanyl required between Group A and Group B

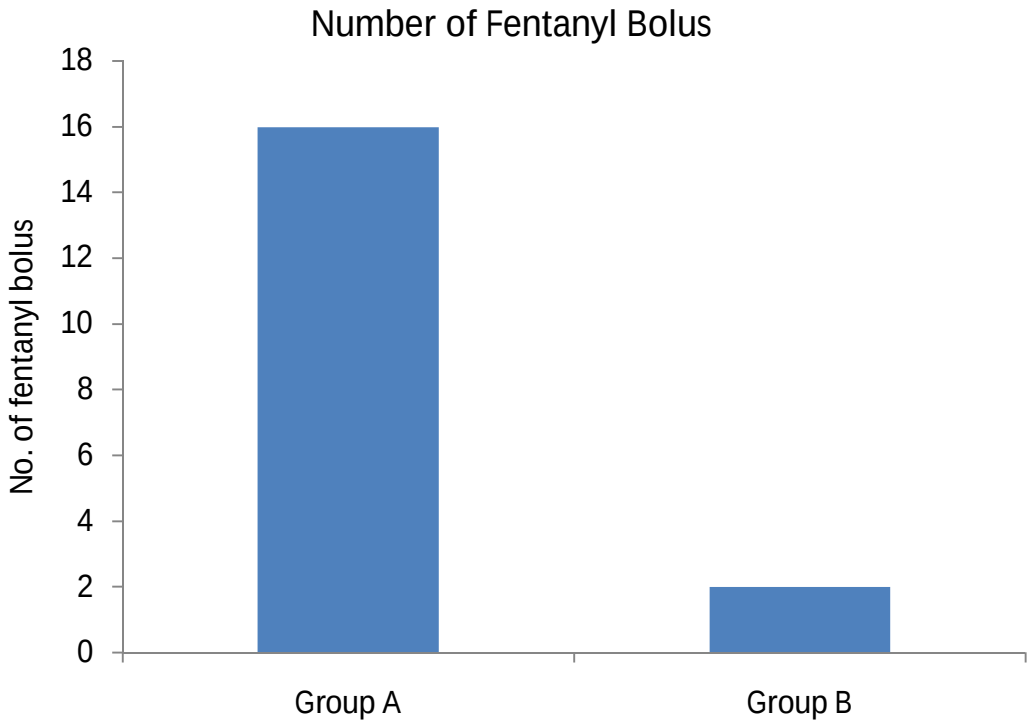
	Group A	Group B	P value
Number of Fentanyl bolus doses	16	2	0.001

The total number of bolus doses of fentanyl required in Group A was 16.

The total number of bolus doses of fentanyl required in Group B was 2.

On analysing the data statistically, the p value was calculated as p=0.001 for total number of bolus doses of fentanyl required in Group A and Group B. All these values were <0.05, hence the difference was statistically significant. Hence we concluded that the number of bolus doses of fentanyl required in group A was significantly higher when compared to group B.

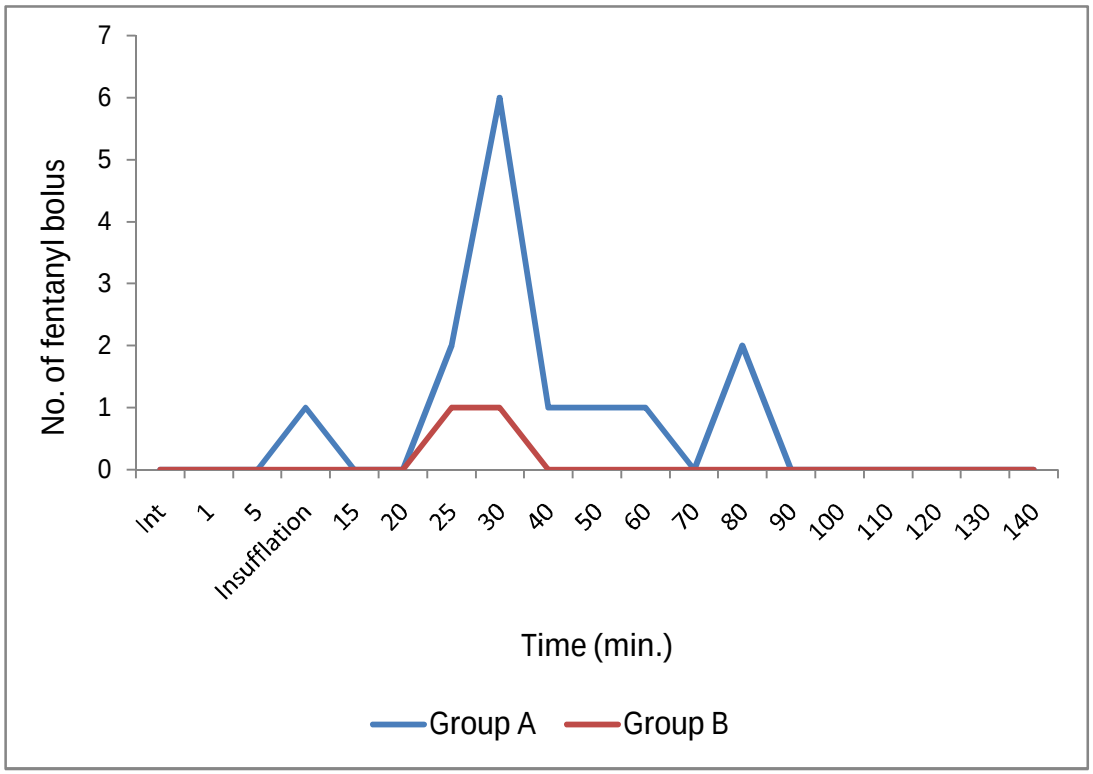
Figure 9: Bar diagram comparing the number of total bolus doses of fentanyl required between Group A and Group B



The total number of bolus doses of fentanyl required in Group A was 16 and in Group B was 2.

Hence the total number of bolus doses of fentanyl required in Group A was significantly higher when compared to Group B.

Figure 10: Comparison of number of fentanyl bolus requirements between Group A and Group B at various time intervals using line diagram



The total number of bolus doses of fentanyl required in Group A was 16 and in Group B was 2. Out of the 16 bolus doses of fentanyl required in Group A, 12 bolus doses were administered within the first 30 minutes of peritoneal insufflation. In Group B both the bolus doses of fentanyl were administered within the first 20 minutes of peritoneal insufflation.

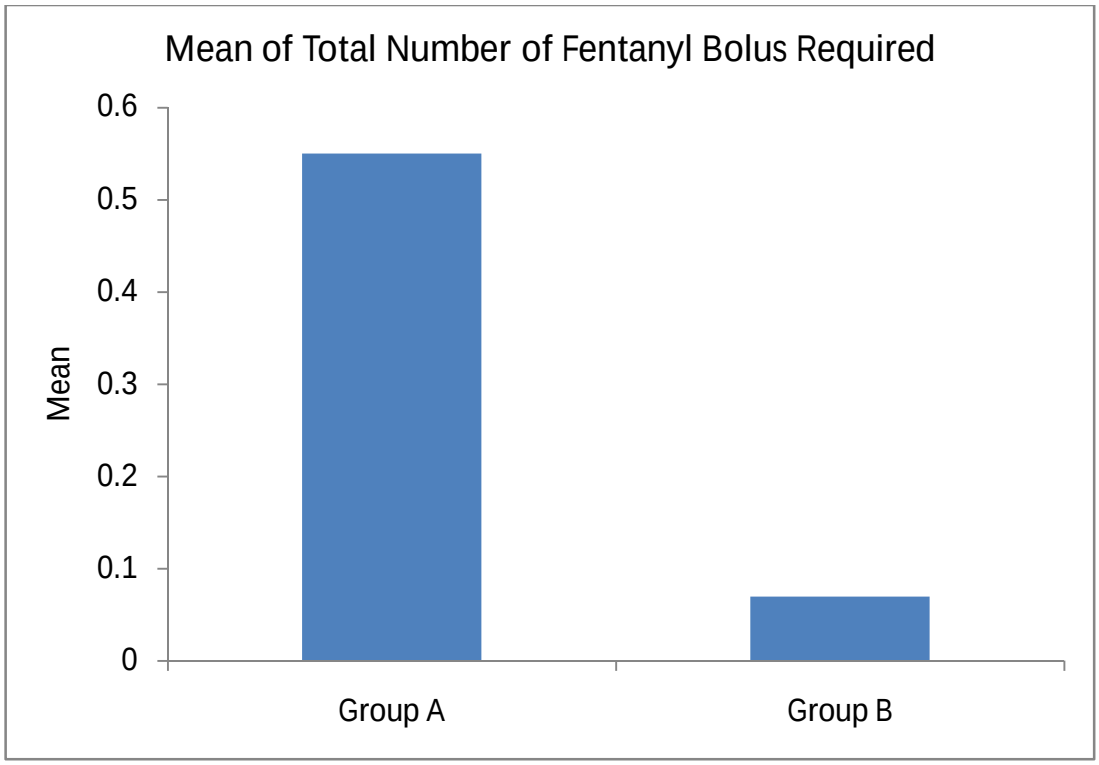
Table 5: Comparison of mean of total number of bolus doses of fentanyl required between Group A and Group B

	Group A	Group B	P value
Bolus doses of Fentanyl	0.55	0.07	0.001

The mean of total number of bolus doses of fentanyl required in Group A was 0.55 while in Group B the mean was 0.07.

On analysing the data statistically, the p value was calculated as p=0.001 for the mean of total number of bolus doses of fentanyl required in Group A and Group B. All these values were <0.05, hence the difference was statistically significant. Hence we concluded that the mean of total number of bolus doses of fentanyl required in group A was significantly higher when compared to group B.

Figure 11: Bar diagram comparing the number of total bolus doses of fentanyl required between Group A and Group B



The mean of total number of bolus doses of fentanyl required in Group A was 0.55.

The mean of total number of bolus doses of fentanyl required in Group B was 0.07.

Table 6: Comparison of total number of episodes of hypotension between Group A and Group B

	Group A	Group B	P value
Number of Episodes of Hypotension	1	23	0.000

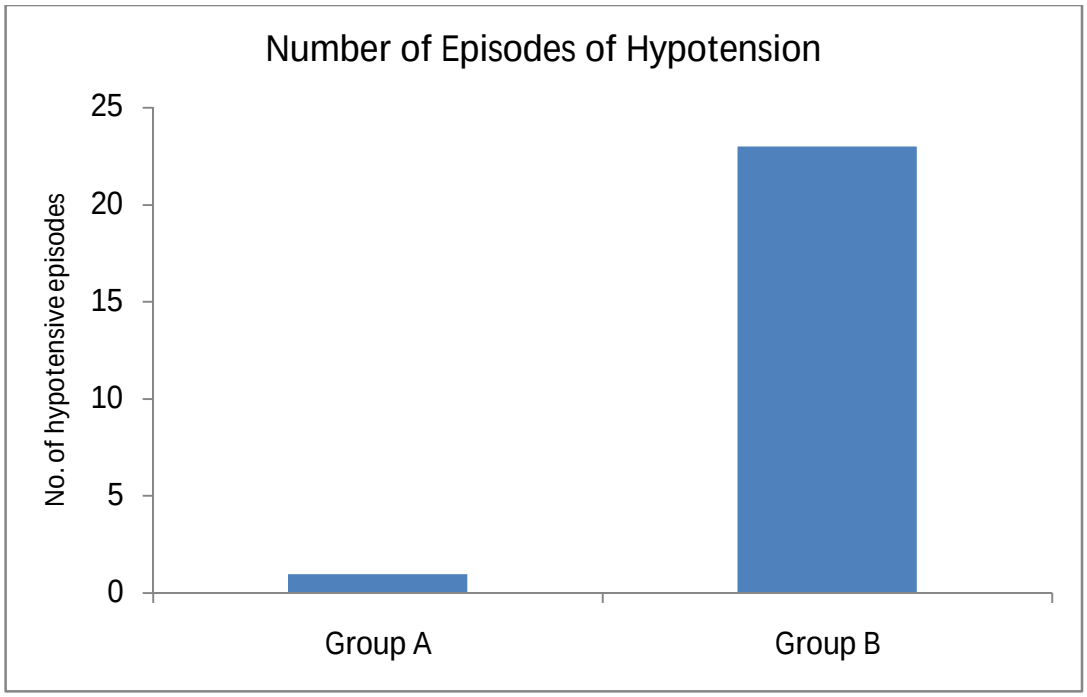
The total number of episodes of hypotension encountered in Group A was 1.

The total number of episodes of hypotension encountered in Group B was 23.

On analysing the data statistically, the p value was calculated as p=0.000 for the total number of episodes of hypotension between Group A and Group B. All these values were <0.05, hence the difference was statistically significant. Hence, the total number of hypotensive episodes was significantly higher in group B when compared to group A.

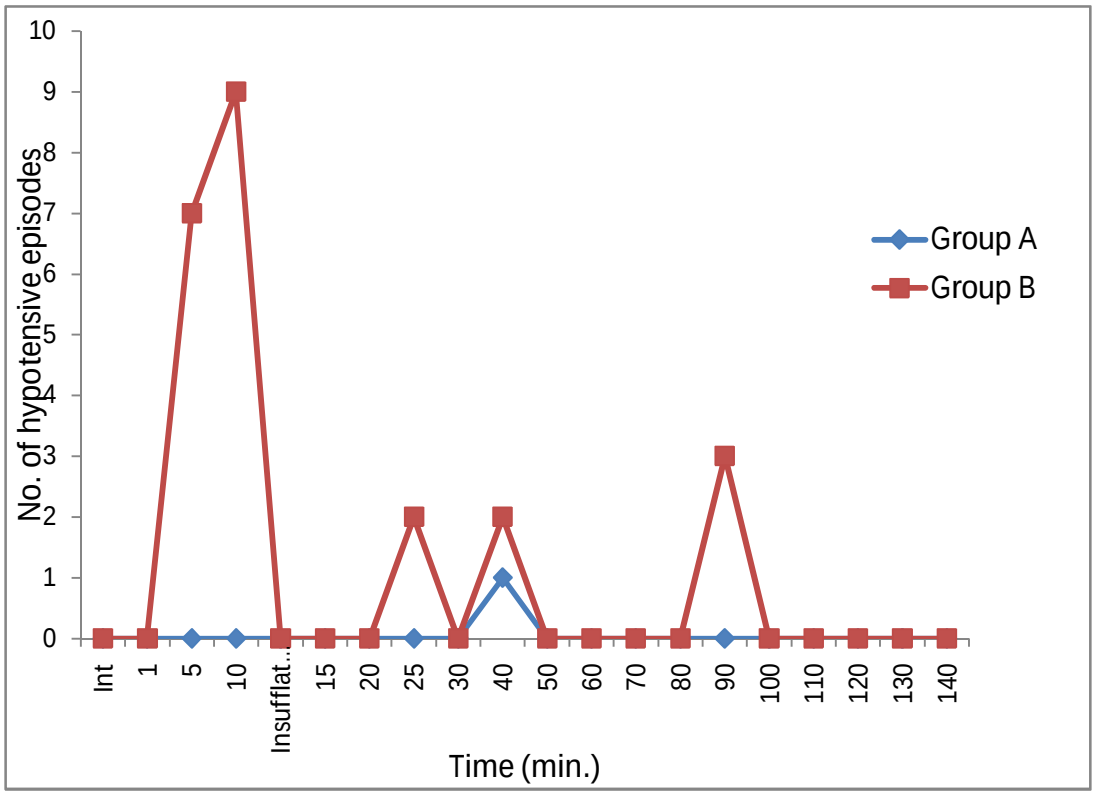
The study protocol defined hypotension as MAP <25% of baseline values on two consecutive readings within 2 – 3 minutes.

Figure 12: Bar diagram comparing the total number of episodes of hypotension between Group A and Group B



The total number of episodes of hypotension encountered in Group A was 1 and in Group B was 23. Hence the number of episodes of hypotension in Group B was significantly higher when compared to Group A.

Figure 13: Comparison of number of episodes of hypotension between Group A and Group B at various time intervals using line diagram



The total number of hypotensive episodes encountered in Group B was 23. 16 of these 23 episodes were observed around 10 minutes following intubation and before peritoneal insufflation. These hypotensive episodes were transient and MAP returned to within 25% of baseline values within 5 minutes of decrease in volatile concentration and administration of a fluid bolus.

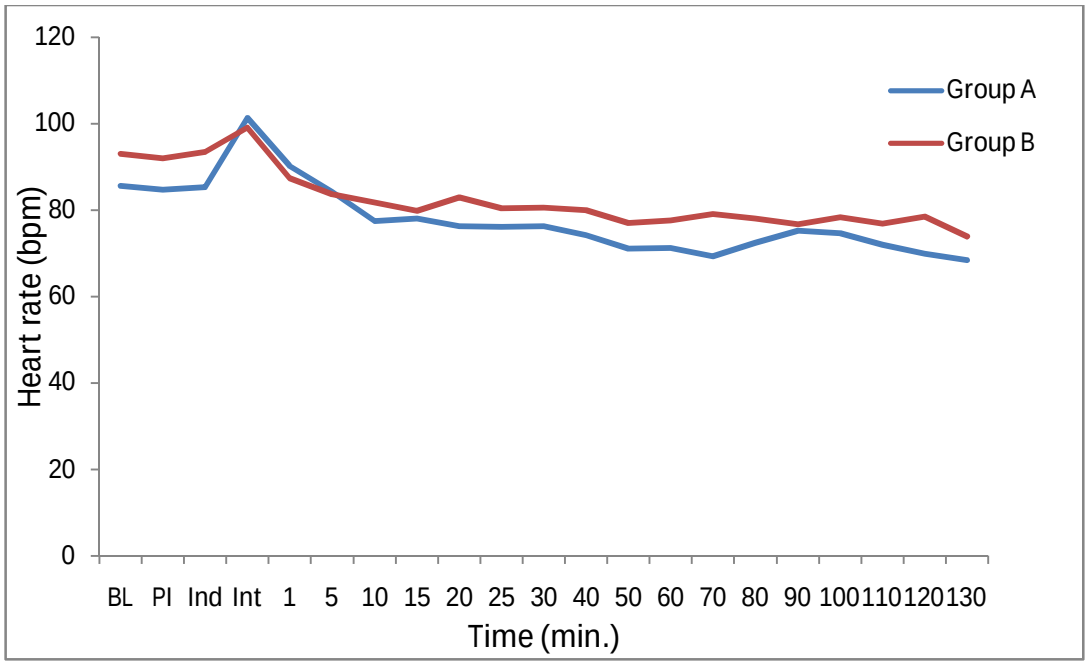
Table 7: Comparison of heart rates between Group A and Group B

HEART RATE

TIME INTERVAL	GROUP A	GROUP B	P VALUE
Baseline	85.72	92.83	.073
Preinduction	84.86	91.03	.076
Induction	85.41	92.90	.058
Intubation	101.38	98.77	.665
1 min	90.28	87.13	.351
5 min	84.31	83.23	.539
10 min	77.48	81.03	.534
15 min	78.14	79.20	.982
20 min	76.28	82.40	.100
25 min	76.24	80.07	.400
30 min	76.31	80.30	.421

TIME INTERVAL	GROUP A	GROUP B	P VALUE
40 min	74.24	79.50	.094
50 min	71.14	76.77	.093
60 min	73.66	77.27	.291
70 min	69.45	78.20	.008
80 min	72.56	77.24	.092
90 min	75.38	76.11	.762
100 min	74.71	76.72	.518
110 min	72.00	75.17	.605
120 min	70.00	78.50	.091
130 min	68.50	74.00	.519

Figure 14: Comparison of heart rate variations between Group A and Group B using line diagram



Maximal increase in heart rate in both the groups occurred following laryngoscopy and endotracheal intubation. The increase in mean heart rate in Group A was from 86 to 101 beats per minute while in Group B mean heart rate increased from 93 to 99 beats per minute during endotracheal intubation. But the increase was not statistically significant and heart rates in both the groups remained within 25 % of baseline values throughout the course of the study.

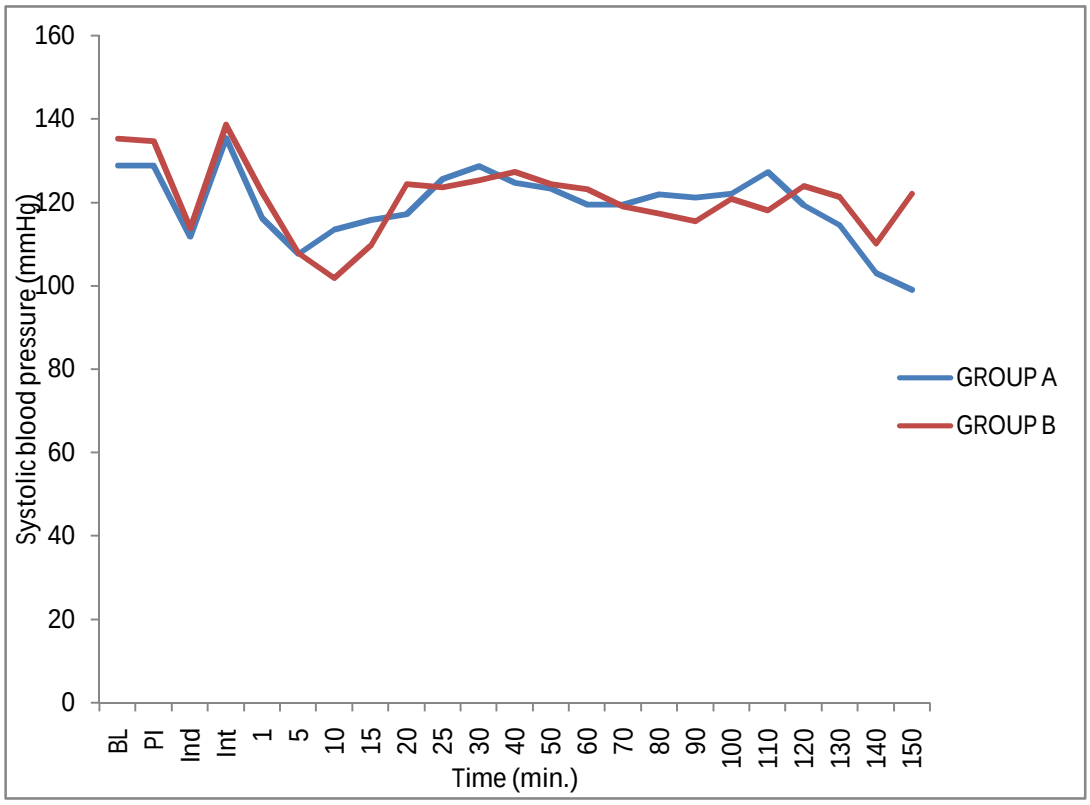
None of the patients in both the groups encountered bradycardia (HR<50) both during intraoperative and postoperative periods.

Table 8: Comparison of systolic blood pressure (mmHg) between Group A and Group B

TIME INTERVAL	GROUP A	GROUP B	P VALUE
Baseline	128.79	135.30	.135
Preinduction	128.76	134.60	.170
Induction	111.79	113.73	.500
Intubation	135.34	138.60	.439
1 min	116.10	122.53	.292
5 min	107.52	107.80	.773
10 min	113.38	101.77	.001
15 min	115.76	109.53	.262
20 min	117.14	124.37	.121
25 min	125.59	123.62	.624
30 min	128.52	125.23	.514
40 min	124.66	127.23	.727

TIME INTERVAL	GROUP A	GROUP B	P VALUE
50 min	123.24	124.30	.575
60 min	119.41	123.13	.379
70 min	119.41	118.40	.982
80 min	121.81	117.28	.358
90 min	121.05	115.46	.073
100 min	121.93	120.78	.901
110 min	127.17	118.08	.244
120 min	119.25	123.85	.648
130 min	114.50	121.33	1.000

Figure 15: Comparison of systolic blood pressure between Group A and Group B at various time intervals using a line diagram



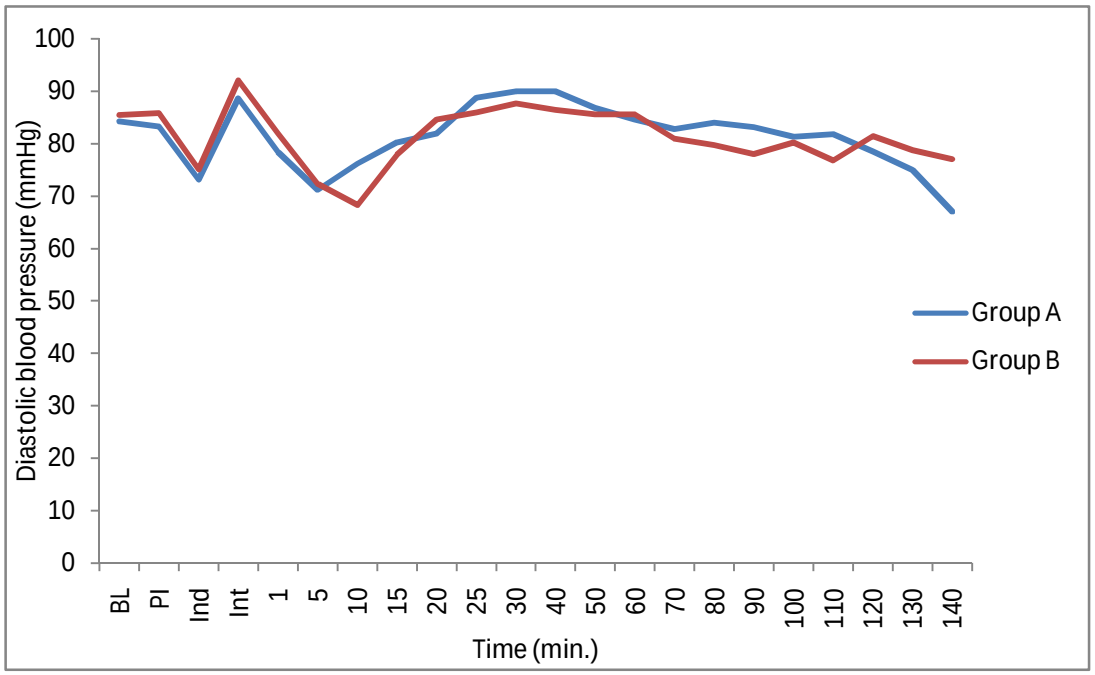
Maximal increase in SBP in both the groups occurred following endotracheal intubation. But the values remained within 25 % of the baseline values and the increase was found not to be statistically significant. However, 19 patients in Group B encountered episodes of fall in SBP to < 25% of baseline values (on two consecutive readings within 2 – 3 minutes) around 10 minutes following tracheal intubation prior to peritoneal insufflation. In Group A only one patient encountered an episode of fall in SBP to < 25% of baseline values.

Table 9: Comparison of diastolic blood pressure (mmHg) between Group A and Group B

TIME INTERVAL	GROUP A	GROUP B	P value
Baseline	84.28	85.47	.569
Preinduction	83.38	85.8	.194
Induction	73.1	75.03	.688
Intubation	88.66	92.1	.387
1 min	78.38	82	.061
5 min	71.14	72.3	.796
10 min	76.17	68.23	.021
15 min	80.24	77.93	.616
20 min	82.03	84.6	.216
25 min	88.83	86	.554
30 min	90.07	87.73	.688
40 min	90.03	86.47	.391

TIME INTERVAL	GROUP A	GROUP B	P value
50 min	86.83	85.67	.998
60 min	84.72	85.67	.761
70 min	82.83	80.93	.791
80 min	84.11	79.79	.651
90 min	83.14	78.04	.073
100 min	81.33	80.21	.758
110 min	81.8	76.85	.289
120 min	78.5	81.43	.527
130 min	75	78.75	1.000

Figure 16: Comparison of diastolic blood pressure between Group A and Group B at various time intervals using line diagram



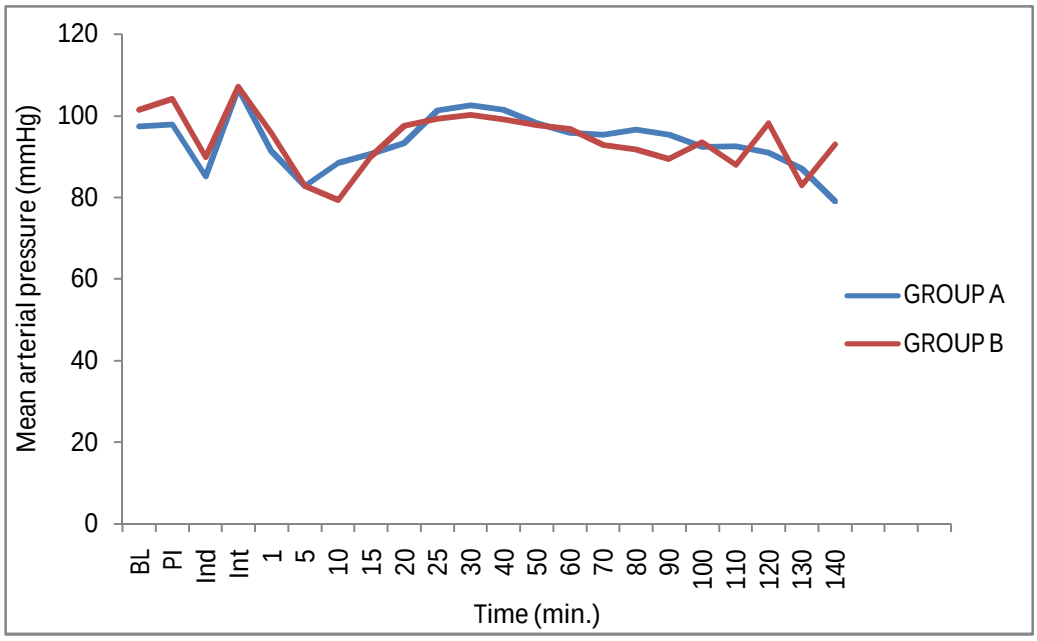
Maximal increase in DBP in both the groups occurred following endotracheal intubation. But the values remained within 25 % of the baseline values and the increase was found not to be statistically significant. However, 19 patients in Group B encountered episodes of fall in DBP to less 25% of baseline values (on two consecutive readings within 2 – 3 minutes) around 10 minutes following tracheal intubation prior to peritoneal insufflation. In Group A only one patient encountered an episode of fall in DBP to less than 25% of baseline values. These episodes of hypotension were transient and corrected by the administration of a fluid bolus and a decrease in volatile concentration.

Table 10: Comparison of mean arterial pressure (mmHg) between Group A and Group B

TIME INTERVAL	GROUP A	GROUP B	P VALUE
Baseline	97.45	101.5	.197
Preinduction	97.73	104.2	.085
Induction	85.17	89.97	.177
Intubation	106.83	107.23	.802
1 min	91.24	95.77	.049
5 min	82.72	82.87	.832
10 min	88.38	79.37	.007
15 min	90.59	90.1	.688
20 min	93.28	97.6	.118
25 min	101.28	99.43	.933
30 min	102.66	100.3	.791
40 min	101.52	99.17	.721

TIME INTERVAL	GROUP A	GROUP B	P VALUE
50 min	98.17	97.73	.676
60 min	95.86	96.87	.756
70 min	95.29	92.93	.640
80 min	96.63	91.86	.485
90 min	95.33	89.43	.023
100 min	92.33	93.61	.929
110 min	92.5	88	.467
120 min	91	98.33	.381
130 min	87	83	1.000

Figure 17: Comparison of mean arterial pressure between Group A and Group B at various time intervals using line diagram



Maximal increase in MAP occurred following endotracheal intubation. The mean rise in MAP was from 98 mmHg to 107 mmHg in Group A while in Group B mean MAP values increased from 93mmHg to 99mmHg. But the values remained within 25% of baseline values and the increase found was not statistically significant. 16 patients in Group A and 2 patients in Group B encountered episodes of hypertension (MAP >25 % on two consecutive readings taken within 2 – 3min). Majority of these episodes of hypertension in both the groups were encountered within the first 30 minutes of peritoneal insufflation.

Maximal decrease in MAP was observed around 10 minutes following tracheal intubation. Episodes of hypotension (MAP<25% of baseline values for two consecutive readings within 2-3 minutes) occurred in 19 patients in Group B while in Group A only one patient encountered an episode of hypotension.

Table 11: Comparison of the mean duration to recovery (eye opening) and post operative sedation scores between Group A and Group B

	Group A	Group B	P value
Duration to eye opening	14.41±3.11	16.87±1.17	0.000
Sedation score	2.00±0.00	2.17±0.38	0.023

The mean duration to eye opening in Group A and Group B was 14.41 and 16.87 minutes respectively.

The mean post-extubation sedation score in Group A was 2 and in Group B was 2.17.

On analysing the data statistically, the p value is calculated was p=0.000, and p=0.023 for duration to eye opening and post operative sedation scores respectively. All these values were <0.05, hence the difference was statistically significant between the two groups in terms of duration to recovery and post operative sedation scores. Hence we concluded that Dexmedetomidine at a dose of 0.6 µg/kg/hr results in an increased post-extubation sedation score and prolongs the duration to eye opening.

CHAPTER 6

DISCUSSION

Laparoscopic surgeries under general anaesthesia are associated with unique hemodynamic changes in the form of decreased venous return and increased systemic vascular resistance leading to systemic hypertension. This increases the need for deepening the plane of anaesthesia and requires the use of vasodilators to counteract the rising blood pressures .IAPs higher than 10 mm Hg due to peritoneal insufflation with CO_2 induces significant alterations in hemodynamics, characterised by decrease in venous return ,increase in arterial pressures ,elevation of systemic and pulmonary vascular resistance .Heart rate remains unchanged or increases only slightly .These hemodynamic perturbations occur mainly at the beginning of peritoneal insufflation.

Various studies have been conducted with different pharmacological interventions that result in reduced incidence of tachycardia, hypertension during laparoscopic cholecystectomy and provide a stable hemodynamic state with minimal undesirable effects.

Dexmedetomidine is a highly selective α_2 adrenoceptor agonist which possesses analgesic , hypnotic, sedative, amnesic, anxiolytic and sympatholytic properties without producing significant respiratory depression. Its sympatholytic effect decreases mean arterial

pressure(MAP) and heart rate by reducing norepinephrine release .In addition Dexmedetomidine has the ability to reduce both the anaesthetic and opioid analgesic requirements during the perioperative period .It differs from clonidine in being 16 times more specific for α_2 receptors . Dexmedetomidine due to such distinct properties can be used as an anaesthetic adjuvant in the form of intravenous infusion for laparoscopic surgeries.

This prospective randomized, double-blind study was conducted in an attempt to compare and examine whether the administration of two different doses of Dexmedetomidine to a commonly administered balanced anaesthetic regimen reduces the perioperative analgesic and volatile requirement. In this prospectively randomized, double-blind study, we tested the hypothesis that Dexmedetomidine infusion would produce dose related reductions in the anaesthetic and analgesic requirements in patients undergoing laparoscopic cholecystectomy. Previously various studies have evaluated different bolus doses of Dexmedetomidine for premedication; however, dose-ranging studies are essential when the drug is administered as a continuous infusion during surgery. Dexmedetomidine has a high propensity to produce hypotension and / or bradycardia and hence it is important to determine an infusion rate that would maximize the anaesthetic and analgesic-sparing effect while minimizing the occurrence of adverse cardiovascular side effects requiring therapeutic interventions. The

addition of initial loading bolus dose of Dexmedetomidine resulted in a high incidence of hypotension immediately following tracheal intubation in a “pilot” experience before initiating the current protocol. Hence we selected two doses of Dexmedetomidine at a constant infusion rate of 0.2 µg/kg/hr and 0.6 µg/kg/hr and compared the anaesthetic and analgesic sparing effects between the two groups. We also compared the efficacy of two doses of Dexmedetomidine on the maintenance of hemodynamic stability in patients undergoing laparoscopic cholecystectomy.

Dexmedetomidine is a potent analgesic and reduces perioperative fentanyl requirements in humans. The α_2 receptors located in the locus ceruleus and the dorsal horn of spinal cord are implicated in the analgesic action of Dexmedetomidine.α_2 agonists and opioids act by diverse mechanisms and their combination provides a synergistic analgesic effect without increasing the incidence of respiratory depression. The initial dose of fentanyl chosen in both groups was 1 µg/kg and bolus doses at 0.5 µg/kg up to a maximum dose of 2 µg/kg in total were supplemented during the course of surgery when warranted as per the study protocol. In our study the requirement of fentanyl was reduced by 20% in the Dexmedetomidine 0.6 group compared to Dexmedetomidine 0.2 group in our study. We also found that a significantly higher number of fentanyl boluses were required in Dexmedetomidine 0.2 group compared with Dexmedetomidine 0.6

group. Therefore in our study we concluded that Dexmedetomidine at a dose of 0.6 µg/kg/hr had better analgesic sparing properties when compared to Dexmedetomidine at 0.2 µg/kg/hr. **Ebert et al**[65] in 2000 studied the hemodynamic responses to increasing concentrations of Dexmedetomidine and concluded that increasing concentrations of Dexmedetomidine resulted in progressive increases in sedation and analgesia . The results of this study are analogous to our study where infusion rate of 0.6µg/kg/hr required significantly lesser amount of fentanyl supplementation suggesting a greater analgesic effect of Dexmedetomidine at a dose of 0.6 µg/kg/hr when compared to the infusion at a rate of 0.2 µg/kg/hr. Our study could not comment on the opioid sparing properties of Dexmedetomidine at dose of 0.2 µg/kg/hr because it is a comparative study and not a controlled study.

It was postulated that a central alpha-2 adrenergic C4 isoreceptor may be involved in the anaesthetic sparing effects of Dexmedetomidine. We chose to compare the desflurane requirements between the two groups and calculated the average inspiratory desflurane concentrations of two groups .The average inspiratory concentrations were calculated as the sum of products of the inspiratory concentrations and times divided by the total anaesthesia time .We found a statistically significant difference in the average inspiratory concentrations between the two groups. The average inspiratory concentration required in group A (Dexmedetomidine 0.2 µg/kg/hr) was 2.66 and the average inspiratory

concentration required in group B (Dexmedetomidine 0.6 µg/kg/hr) was 2.57. Hence it was concluded that Dexmedetomidine at a dose of 0.6 µg/kg/hr has statistically significant anaesthetic sparing effect when compared to Dexmedetomidine at a dose of 0.2 µg/kg/hr. The observations in our study were analogous to the findings of **Feld et al**[62] who combined fentanyl or Dexmedetomidine with desflurane for bariatric surgery and concluded that, desflurane concentrations necessary to maintain bispectral index at 45 to 50 were decreased, and blood pressure and heart rate were decreased with Dexmedetomidine when compared with the fentanyl group. **Anta et al**[64] in another study showed that the MAC of isoflurane was 47 % less with a high dose of Dexmedetomidine than that without Dexmedetomidine. Again due to the lack of a control group, our study could only conclude that Dexmedetomidine decreases the requirements of volatile anaesthetics to a greater degree at a dose of 0.6 µg/kg/hr when compared to a dose of 0.2 µg/kg/hr, but could not comment on the anaesthetic sparing effects of Dexmedetomidine as a whole.

Endotracheal intubation is associated with significant increase in arterial pressure, heart rate, and plasma catecholamine concentrations. Dexmedetomidine attenuated the sympathoadrenal response during tracheal intubation effectively but did not completely abolish the cardiovascular response. There was an increase in both MAP and heart rate in both the groups after intubation .But the values remained within

25 % of the baseline values and the increase found was not statistically significant .The increase in mean heart rate in Dexmedetomidine 0.2 µg/kg/hr group was from 86 bpm to 101 bpm while that in Dexmedetomidine 0.6 µg/kg/hr group was from 93 bpm to 99 bpm during endotracheal intubation. The raise in MAP was from 98 mm Hg to 107 mm Hg in Dexmedetomidine 0.2 µg/kg/hr group while that in Dexmedetomidine 0.6 µg/kg/hr group was from 102 mmHg to 107 mmHg during intubation. Even though the increase in Heart rate and MAP was observed in both the groups during endotracheal intubation, the degree of increase was lesser in Dexmedetomidine 0.6 µg/kg/hr group when compared to the Dexmedetomidine 0.2 µg/kg/hr group. Dexmedetomidine in doses of 0.6 µg/kg/hr had greater sympathoadrenal attenuation properties during endotracheal intubation when compared with doses of 0.2 µg/kg/hr. **Yildiz et al**[77] showed that a preinduction intravenous dose of Dexmedetomidine 1 µg/kg, decreased the need for thiopental and sevoflurane by 39% and 92% respectively, and effectively blunted the hemodynamic responses to laryngoscopy. However our study protocol does not include a preinduction bolus dose of Dexmedetomidine and better attenuation of hemodynamic response to laryngoscopy and intubation were observed in Dexmedetomidine at an infusion rate of 0.6 µg/kg/hr, but none of the groups completely abolished the intubation stress response.

The use of intraoperative Dexmedetomidine may increase the hemodynamic stability due to the attenuation of stress-induced hemodynamic response to intubation, peritoneal insufflation and emergence from anaesthesia. The MAPs were compared at various time intervals between the two groups. Intubation stress response evoked a 10% increase in MAP in Dexmedetomidine 0.2 µg/kg/hr group from the baseline values while Dexmedetomidine 0.6 µg/kg/hr group showed only a 5 % increase in MAP from the baseline values. Mean heart rates between the two groups were compared and Dexmedetomidine 0.2 µg/kg/hr group recorded a mean increase in heart of 20% above baseline value on endotracheal intubation, while the mean increase in Dexmedetomidine 0.6 µg/kg/hr group was 5% above the baseline values . None of the groups encountered a significant increase in heart rate on peritoneal insufflations. None of the patients in both the groups encountered a significant bradycardia with a heart rate of less than 50bpm. Dexmedetomidine at a dose of 0.6µg/kg/hr had better attenuation of sympathoadrenal response to endotracheal intubation when compared to Dexmedetomidine at doses of 0.2 µg/kg/hr, but did not completely obtund the response to endotracheal intubation.

The maximum decrease in MAP in both the groups was observed around 10 minutes following intubation and before peritoneal insufflations . 50 % of patients in Dexmedetomidine 0.6 µg/kg/hr group showed a decrease in MAP greater than 25 % from the baseline values

before peritoneal insufflations , requiring administration of a fluid bolus and a decrease in volatile concentration .However this decrease was transient and MAP values returned to within 25% of baseline values, within 5 minutes of reducing the volatile concentrations and administration of a fluid bolus. None of the patients in Dexmedetomidine 0.2 µg/kg/hr group showed a decrease in MAP greater than 25% from the baseline values before peritoneal insufflations. hroughout the course of the surgery 19 patients in Dexmedetomidine 0.6 µg/kg/hr group required a decrease in volatile and administration of a fluid bolus for correction of hypotension. These hypotensive episodes were transient and MAP returned to within 25% of the baseline values within 5 minutes of decrease in volatile concentration and administration of a fluid bolus. While in Dexmedetomidine 0.2 µg/kg/hr group only 1 patient had a hypotensive episode requiring a fluid bolus and a decrease in volatile concentration. The number of hypotensive episodes encountered in Dexmedetomidine 0.6 µg/kg/hr group were found to be significantly higher when compared to the Dexmedetomidine 0.2 µg/kg/hr group. This could be attributed to the greater sympatholytic effect of Dexmedetomidine at 0.6 µg/kg/hr compared to 0.2 µg/kg/hr. **Ebert et al**[65] in 2000 studied the hemodynamic responses to increasing concentrations of Dexmedetomidine and concluded that increasing concentrations of Dexmedetomidine in humans resulted in progressive increases in

sedation and analgesia and progressive decreases in heart rate, cardiac output and memory. The results of this study are analogous to our study where infusion doses of 0.6 µg/kg/hr produced greater episodes of decrease in MAP when compared to an infusion rate of 0.2 µg/kg/hr.

Hypertensive episodes with MAP values rising to over 25% of baseline values were observed in 15 patients in our study with 13 patients in Dexmedetomidine 0.2 µg/kg/hr group and 2 patients in Dexmedetomidine 0.6 µg/kg/hr group .These hypertensive episodes were treated by supplementing fentanyl boluses in doses of 0.5 µg/kg up to a maximum of 2 µg/kg. All the hypertensive episodes resolved with fentanyl supplementation and none of the cases required an increase in volatile concentration or propofol supplementation in our study .Hypertensive episodes occurred within 30 min of peritoneal insufflations in 8 of the 13 patients in Dexmedetomidine 0.2 µg/kg/hr group .In Dexmedetomidine 0.6 µg/kg/hr group 2 patients who encountered hypertension had these episodes within 20 min of peritoneal insufflations. The number of hypertensive episodes in Dexmedetomidine 0.2 µg/kg/hr group was significantly higher when compared to Dexmedetomidine 0.6 µg/kg/hr group. Out of the 13 patients in Dexmedetomidine 0.2 µg/kg/hr group who encountered intraoperative hypertension, 3 patients required administration of two bolus doses of fentanyl at 0.5 µg/kg to correct hypertension, while in the remaining 10 patients administration of a single bolus dose of fentanyl

at 0.5 µg/kg was sufficient to correct the hypertensive episode. Hypertensive episode that occurred in 2 patients in Dexmedetomidine 0.6 µg/kg/hr group were treated by the administration of a single bolus dose of fentanyl at 0.5 µg/kg. Thus concluding that Dexmedetomidine in doses of 0.6 µg/kg/hr has better sympathoadrenal stress response attenuating properties when compared to Dexmedetomidine at a dose of 0.2 µg/kg/hr. Again the observations in our study are analogous to the study done by **Ebert et al**[65] in 2000 where he showed that progressively increasing concentrations of Dexmedetomidine resulted in progressive increase in sedation and analgesia and progressive decreases in heart rate, cardiac output and memory. Thus Dexmedetomidine at 0.6 µg/kg/hr has better analgesic and sympatholytic properties when compared to Dexmedetomidine at doses of 0.2 µg/kg/hr.

Post extubation sedation scores were studied in the two groups. All patients in Dexmedetomidine 0.2 µg/kg/hr had Ramsay sedation score of 2; whereas 5 patients among the 29 patients in Dexmedetomidine 0.6 µg/kg/hr group had a sedation score of 3. This difference in the post extubation sedation score between the two groups was statistically significant. All patients in both the groups at the end of the second hour following extubation had a sedation score of 2. This was in accordance to the study done by **Ebert et al**[65] who showed that progressively increasing concentrations of Dexmedetomidine result in progressive increase in sedation and analgesia. The mean duration for

eye opening after the cessation of Dexmedetomidine infusion showed a significant increase in Dexmedetomidine 0.6 µg/kg/hr at 16.87min in our study compared to the Dexmedetomidine 0.2 µg/kg/hr group where the mean duration was 14.41 min. Thus Dexmedetomidine at 0.6 µg/kg/hr resulted in a progressive increase in sedation scores and the time for eye opening when compared to Dexmedetomidine at 0.2 µg/kg/hr.

Dexmedetomidine was well tolerated and no serious side effects or adverse reactions occurred both during the intraoperative and post-operative periods in the present study**.**

CONCLUSION

Dexmedetomidine at an infusion rate of 0.6 µg/kg/hr has a better analgesic and anaesthetic sparing property and a better hemodynamic stabilization property when compared to an infusion rate of 0.2 µg/kg/hr, with episodes of transient hypotension, increased postoperative sedation scores, with no serious side effects or adverse reactions.

Hence I conclude that Dexmedetomidine administered at an infusion rate of 0.6 µg/kg/hr may serve as an ideal anaesthetic adjuvant in patients undergoing laparoscopic cholecystectomy.

ANNEXURES

ETHICAL COMMITTEE APPROVAL LETTER

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work	: Dexmedetomidine for Laparoscopic Cholecystectomy- Comparison between two doses on the effects on Sympatho-adrenal response and Anaesthetic requirements.
Principal Investigator	: Dr. R. Praneeth
Designation	: PG in MD (Anaesthesiology)
Department	: Department of Anaesthesiology Government Stanley Medical College, Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.11.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

PROFORMA

NAME:	AGE/SEX:	IP NO.:
DATE:	Wt.:	GROUP:
DIAGNOSIS:	SURGERY:	
BRIEF HISTORY:	COEXISTING ILLNESS:	

EXAMINATION

PR:	CVS:
BP:	RS:
RR:	AIRWAY:

INVESTIGATIONS

Hb:	Blood urea:
Urine alb:	Sugar :
Sr. Creatinine:	Electrolytes:
X-ray chest:	ECG:

ANAESTHESIA DETAILS

Premedication :	Group :
Induction :	Intubation :
Maintenance :	Duration of surgery:

PROFORMA

Parameters	Baseline	Preinduction	Induction	Intubation	1 min	5 min	10 min	15 min	20 min	25 min	30 min	40 min	50 min	60 min	70 min	80 min	90 min	100 min	110 min	120 min	130 min	140 min
HR																						
SBP																						
DBP																						
MAP																						
SPO2																						
ETCO2																						
DESFLUR																						
FENTANYL																						
DEXMED																						

TOTAL FENTANYL :

AVERAGE INSPIRATORY DESFLURANE CONC:

TIME TO EYE OPENING / TRACHEAL EXTUBATION:

INTRA-ABDOMINAL PRESSURE:

TOTAL IV FLUIDS:

BIBLIOGRAPHY

1. Struthers AD, Cuschieri A: Cardiovascular consequences of laparoscopic surgery. Lancet 352:568, 1998.

2. Maze M, Tranquilli W: Alpha-2 adrenoceptor agonists: Defining the role in clinical anaesthesia. Anesthesiology 74:581-605, 1991

3. History of laparoscopic surgery . Panminerva Med. 2000 Mar;42(1):87-90

4. Wahba RW, Tessler MJ, Kleiman SJ: Acute ventilator complications during laparoscopic upper abdominal surgery. Can J Anaesth 43:77, 1996.

5. Bardoczky GI, Engelman E, Levarlet M, et al: Ventilatory effects of pneumoperitoneum monitored with continuous spirometry. Anaesthesia 48:309, 1993

6. Fahy BG, Barnas GM, Flowers JL, et al: The effects of increased abdominal pressure on lung and chest wall mechanics during laparoscopic surgery. Anesth Analg 81:744, 1995.

7. Fahy BG, Barnas GM, Nagle SE, et al: Changes in lung and chest wall properties with abdominal insufflation of carbon dioxide are immediately reversible. Anesth Analg 82:501, 1996.

8. Mullet C, Viale J, Sagnard P, et al: Pulmonary CO2 elimination during surgical procedures using intraor extraperitoneal CO2 insufflation. Anesth Analg 76:622, 1993

9. Whiston RJ, Eggers KA, Morris RW, et al: Tension pneumothorax during laparoscopic cholecystectomy. Br J Surg 78:1325, 1991.

10. Knos GB, Sung YF, Toledo A: Pneumopericardium associated with laparoscopy. J Clin Anesth 3:56, 1991

11. Joris JL, Chiche JD, Lamy ML: Pneumothorax during laparoscopic fundoplication: diagnosis and treatment with positive end-expiratory pressure. Anesth Analg 81:993, 1995.

12. *Wahba RW, Tessler MJ, Kleiman SJ: Acute ventilatory complications during laparoscopic upper abdominal surgery. Can J Anaesth 43:77,* 1996.

13. Bottcher-Haberzeth S, Dullenkopf A, Gitzelmann CA, Weiss M: Tracheal tube tip displacement during laparoscopy in children. Anaesthesia 62:131, 2007

14. McGrath BJ, Zimmerman JE, Williams JF, et al: Carbon dioxide embolism treated with hyperbaric oxygen. Can J Anaesth 36:586, 1989.

15. Giebler RM, Behrends M, Steffens T, et al: Intraperitoneal and retroperitoneal carbon dioxide insufflations evoke different effects on caval vein pressure gradients in humans: Evidence for the starling resistor concept of abdominal venous return. Anesthesiology 92:1568, 2000

16. Kashtan J, Green JF, Parsons EQ, et al: Hemodynamic effect of increased abdominal pressure. J Surg Res 30:249, 1981.

17. Cunningham AJ, Turner J, Rosenbaum S, Rafferty T: Transoesophageal echocardiographic assessment of haemodynamic function during laparoscopic cholecystectomy. Br J Anaesth 70:621, 1993

18. Wahba RW, Beique F, Kleiman SJ: Cardiopulmonary function and laparoscopic cholecystectomy. Can J Anaesth 42:51, 1995.

19. Longhurst JC, Spilker HL, Ordway GA: Cardiovascular reflexes elicited by passive gastric distension in anesthetized cats. Am J Physiol 240:H539, 1981

20. Alishahi S, Francis N, Crofts S, et al: Central and peripheral adverse hemodynamic changes during laparoscopic surgery and their reversal with a novel intermittent sequential pneumatic compression device. Ann Surg 233:176, 2001.

21. Iwase K, Takenaka H, Ishizaka T, et al: Serial changes in renal function during laparoscopic cholecystectomy. Eur Surg Res 25:203, 1993.

22. Fujii Y, Tanaka H, Tsuruoka S, et al: Middle cerebral arterial blood flow velocity increases during laparoscopic cholecystectomy. Anesth Analg 78:80, 1994

23. Brantley JC 3rd, Riley PM: Cardiovascular collapse during laparoscopy: A report of two cases. Am J Obstet Gynecol 159:735, 1988.

24. Hirvonen EA, Nuutinen LS, Kauko M: Hemodynamic changes due to Trendelenburg positioning and pneumoperitoneum during laparoscopic hysterectomy. Acta Anaesthesiol Scand 39:949, 1995.

25. Bazin JE, Gillart T, Rasson P, et al: Haemodynamic conditions enhancing gas embolism after venous injury during laparoscopy: a study in pigs. Br J Anaesth 78:570, 1997.

26. Odeberg S, Ljungqvist O, Svenberg T, et al: Haemodynamic effects of pneumoperitoneum and the influence of posture during anaesthesia for laparoscopic surgery. Acta Anaesthesiol Scand 38:276,

27. Karayiannakis AJ, Makri GG, Mantzioka A, et al: Systemic stress response after laparoscopic or open cholecystectomy: A randomized trial. Br J Surg 84:467, 1997

28. .Senagore AJ, Kilbride MJ, Luchtefeld MA, et al: Superior nitrogen balance after laparoscopicassisted colectomy. Ann Surg 221:171, 1995.

29. Rademaker BM, Ringers J, Odoom JA, et al: Pulmonary function and stress response after laparoscopic cholecystectomy: Comparison with subcostal incision and influence of thoracic epidural analgesia. Anesth Analg 75:381, 1992

30. Aho M, Scheinin M, Lehtinen AM, et al: Intramuscularly administered dexmedetomidine attenuates hemodynamic and stress hormone responses to gynecologic laparoscopy. Anesth Analg 75:932, 1992.

31. Fredman B, Jedeikin R, Olsfanger D, et al: Residual pneumoperitoneum: A cause of postoperative pain after laparoscopic cholecystectomy. Anesth Analg 79:152, 1994.

32. Erice F, Fox GS, Salib YM, et al: Diaphragmatic function before and after laparoscopic cholecystectomy. Anesthesiology 79:966, 1993. 196. Sharma RR, Axelsson H, Oberg A, et

33. Civantos CB, de Artinano AA, Alpha adrenoceptor subtypes. Pharmacological Research.2001;44(3):195-208

34. Ahlquist RP. A Study of Adrenotropic receptors. Am J Physiol. 1948; 153(3):586-600

35. Laner SZ .Presynaptic regulation of catecholamine release.Biochemical pharmacology.1974;23:1793-800

36. Bylund DB. Heterogeneity of alpha 2 adrehergic receptors. Pharmacology, Biochemistry & Behavior. 1985; 22(5): 835-43

37. Aantaa R ,Marjamaki A,Scheinin M .Molecular pharmacology of alpha-2 adrenoceptor subtypes. Annals of Medicine. 1995; 27(4): 439-49

38. Codina J,Yatani A, Grenet D, Brown AM, Birnbaumer L. The alpha subunit of the GTP binding protein GK opens atrial potassium channels. Science. 1987; 236(4800): 442-5.

39. Unnerstall JR, Kopajtic TA, Kuhar MJ. Distribution of alpha 2 agonist binding sites in the rat and human central nervous system :analysis of some functional, anatomic correlates of the pharmacologic effects of Clonidine and related adrenergic agents. Brain Res. 1984; 319(1): 69-101.

40. Kamibayashi T, Maze M. Clinical uses of alpha 2 adrenergic agonists. Anesthesiology. 2000;93(5): 1345-9.

41. U.S.Food and Drug Administration. Precedex prescribing information.cited October 2010.

42. Venn RM, Karol MD, Grounds RM. Pharmacokinetics of Dexmedetomidine infusions for sedation of postoperative patients requiring intensive care. British Journal of Anaesthesia. 2002; 88(5): 669-75.

43. Karol MD, Maze M. Pharmacokinetics and interaction Pharmacodynamics of Dexmedetomidine in Humans. Best Practice & Research Clinical Anaesthesiology. 2000; 14(2): 261-9

44. Panzer O, Moitra V, Sladen RN. Pharmacology of sedative –analgesic agents: Dexmedetomidine, Remifentanil, Ketamine, volatile anaesthetics, and the role of peripheral mu agonists. Anesthesiology Clin. 2011; 29:587-605

45. Davies MF, Haimor F, Lighthall G, Clark JD: Dexmedetomidine fails to cause hyperalgesia after cessation of chronic administration. Anesth Analg 96:195-200, 2003.

46. Aho M, Erkola O, Kallio A, et al: Comparison of dexmedetomidine and midazolam sedation and antagonism of dexmedetomidine with atipamezole. J Clin Anesth 5:194-203, 1993

47. Kuhmonen J, Pokorny J, Miettinen R, et al: Neuroprotective effects of dexmedetomidine in the gerbil hippocampus after transient global ischemia. Anesthesiology 87:371-377, 1997.

48. Engelhard K, Werner C, Kaspar S, et al: Effect of the alpha2-agonist dexmedetomidine on cerebral neurotransmitter concentrations during cerebral ischemia in rats. Anesthesiology 96:450-457, 2002.

49. Talke P, Bickler PE: Effects of dexmedetomidine on hypoxia-evoked glutamate release and glutamate receptor activity in hippocampal slices. Anesthesiology 85:551-557, 1996.

50. Asano T, Dohi S, Ohta S, et al: Antinociception by epidural and systemic alpha(2)-adrenoceptor agonists and their binding affinity in rat spinal cord and brain. Anesth Analg 90:400-407, 2000

51. Kaya FN, Yavascaoglu B, Turker G, Yildirim A, Gurbet A, Mogol EB, Ozcan B: Intravenous dexmedetomidine, but not midazolam, prolongs bupivacaine spinal anaesthesia. Can J Anaesth 2010, 57:39-45.

52. Rutkowska K, Knapik P, Misiolek H: The effect of dexmedetomidine sedation on brachial plexus block in patients with endstage renal disease. Eur J Anaesthesiol 2009, 26:851-5.

53. Al-Zaben KR, Qudaisat IY, Al-Ghanem SM, Massad IM, Al- Mustafa MM, Al-Oweidi AS, Abu-Halaweh SA, Abu-Ali HM, Saleem MM: Intraoperative administration of dexmedetomidine reduces the analgesic requirements for children undergoing hypospadius surgery. Eur J Anaesthesiol 2010, 27:247-52.

54. Unlugenc H, Gunduz M, Guler T, Yagmur O, Isik G: The effect of pre-anaesthetic administration of intravenous dexmedetomidine on postoperative pain in patients receiving patientcontrolled morphine. Eur J Anaesthesiol 2005, 22:386-91.

55. Yung-Wei H, Robertson K, Young C, et al: Compare the respiratory effects of remifentanil and dexmedetomidine.Anesthesiology 95:A1357, 2001.

56. Roekaerts P, Prinzen F, Willingers H. The effects of Dexmedetomidine on Systemic Hemodynamics , regional myocardial function and blood flow during coronary artery stenosis in acute anesthetised dogs. J Cardiothorac Anesth.1994;8:58

57. Venn M, Newman J, Grouns M. A phase II Study to evaluate the effects of Dexmedetomidine for sedation in the medical intensive care unit.Intensive Care Med.2003;29:201-207

58. Triltsch AE, Welte M, von Homeyer P, et al: Bispectral index-guided sedation with dexmedetomidine in intensive care: a prospective, randomized, double blind, placebo-controlled phase II study. Crit CareMed 30:1007-1014, 2002.

59. Maccioli GA: Dexmedetomidine to facilitate drug withdrawal. Anesthesiology 98:575-577, 2003.

60. Aantaa R, Kanto J, Scheinin M, et al: Dexmedetomidine, an alpha 2-adrenoceptor agonist, reduces anaesthetic requirements for patients undergoing minor gynecologic surgery. Anesthesiology 73:230- 235, 1990.

61. Aho M, Lehtinen AM, Erkola O, et al: The effect of intravenously administered dexmedetomidine on perioperative hemodynamics and

isoflurane requirements in patients undergoing abdominal hysterectomy. Anesthesiology 74:997-1002, 1991.

62. Feld JM, Hoffman WE, Stechert MM, et al: Fentanyl or dexmedetomidine combined with desflurane for bariatric surgery. J Clin Anesth 18:24-28, 2006.

63. Dholakia C, Beverstein G, Garren M, et al: The impact of perioperative dexmedetomidine infusion on postoperative narcotic use and duration of stay after laparoscopic bariatric surgery. J Gastrointest Surg 11:1556-1559, 2007.

64. Aantaa R, Jaakola ML, Kallio A, Kanto J. Reduction of the minimum alveolar concentration of isoflurane by dexmedetomidine.Anesthesiology 1997;86:1055–60

65. Hall Judith E , Uhrich Toni D , Barney Jill A , Colinco Maelynn ,Ebert Thomas J.The effects of increasing plasma concentrations of Dexmedetomidine in Humans. Anesthesiology. 2000;93:382–94

66. Aho M, Lehtinen AM, Erkola O, Kallio A, Korttila K. The effect of intravenously administered dexmedetomidine on perioperative hemodynamics and isoflurane requirements in patients undergoing abdominal hysterectomy. Anesthesiology 199;74:997–1002

67. Feld JM, Hoffman WE, Stechert MM, Hoffman IW, Ananda RC. Fentanyl or dexmedetomidine combined with desflurane forbariatric surgery. J Clin Anesth 2006;18:24–8

68. Hall Judith E , Uhrich Toni D , Barney Jill A , Arain Shahbaz R ,Ebert Thomas J. Sedative ,Amnestic and Analgesic properties of small doses of Dexmedetomidine . Anesth Analg 2000;90:699-705

69. Aho M, Erkola O, Kallio A, Scheinin H, Korttila K. Dexmedetomidine infusion for maintenance of anaesthesia in patients undergoing abdominal hysterectomy. Anesth Analg 1992;75:940 – 6

70. Talke P, Richardson CA, Scheinin M, Fisher DM. Postoperative pharmacokinetics and sympatholytic effects of dexmedetomidine. Anesth Analg. 1997 Nov;85(5):1136-42

71. Arain SR, Ebert TJ. The efficacy, side effects, and recovery characteristics of dexmedetomidine versus propofol when usedfor intraoperative sedation. Anesth Analg 2002;95:461–6

72. Gurbet A, Basagan-Mogol E, Turker G, Ugun F, Kaya FN, Ozcan B. Intraoperative infusion of dexmedetomidine reduces perioperative analgesic requirements. Can J Anaesth 2006;53:646 –52

73. Talke P, Li J, Jain U, Leung J, Drasner K, Hollenberg M, Mangano DT. Effects of perioperative dexmedetomidine infusion in patients undergoing vascular surgery. The Study of Perioperative Ischemia Research Group. Anesthesiology 1995;82:620–3

74. Dexmedetomidine Infusion During Laparoscopic Bariatric Surgery: The Effect on Recovery Outcome Variables. Burcu Tufanogullari et al. Anesth Analg 2008;106:1741–8

75. Hirvonen EA , Poikolainen EO , Paakkonen ME, Nuutinen LS. The adverse hemodynamic effects of anaesthesia, head-up tilt, and carbon dioxide pneumoperitoneum during laparoscopic cholecystectomy . Surg Endosc 2000; 14(3) : 272-7

76. Jorris J,Noirot D, Legrand MJ, Jacquet NJ, Lamy ML. Hemodynamic Changes during Laparoscopic Cholecystectomy. Anesth Analg 1993; 76: 1067-72

77. Effect of dexmedetomidine on haemodynamic responses to laryngoscopy and intubation : perioperative haemodynamics and anaesthetic requirements. Yildiz M1, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu S.Drugs R D. 2006;7(1):43-52

INFORMATION SHEET

1. You have been accepted.

2. We are conducting a study on "Dexmedetomidine for Laparoscopic Cholecystectomy – Comparison between two doses on the effects on sympathoadrenal response and anaesthetic sparing properties", Stanley Medical College, Chennai and for that you may be valuable to us.

3. We are selecting certain patients and if you are found eligible, we may be using you to perform procedures which will not harm you.

4. The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

5. Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

Date:

Signature of Investigator Signature of Participant

நோயாளி தகவல் தாள்

லாப்ரோஸ்கொபி கருவியின் துணைகொண்டு பித்தப்பை அகற்று அறுவை சிகிச்சை மேற்கொள்ளும் பொழுது கொடுக்கபடும் முழுமயக்கத்தோடு டெக்ஸ்மெடெடொமிடின் என்ற மருந்தை இருவேறு அளவில் இரத்தானானதில் அளித்து அதனால் உடலில் ஏற்படும் இரத்த அழுத்தம் , நாடி துடிப்பு , மற்ற மயக்க மருந்துகளின் தேவை ஆகியவற்றின் ஒப்பிடும் ஆய்வு.

ஆராய்ச்சியின் நோக்கமும் , ஆதரங்களும் :

அறுவை சிகிச்சை செய்வதற்கு மயக்க மருந்து மிகவும் அவசியமானது . அவ்வாறான மயக்க மருந்துகளும் மயக்க முறைகளும் பல வகை உண்டு. பித்தப்பை அறுவை சிகிச்சைக்கு வாய்வழியாக ட்யூப் செலுத்தி செயற்கை சுவாசம் கொடுக்கும் முறை அப்பொழுது பயன்படுத்தபடும் நோயாளி மயக்கநிலயில் இருக்க பல மருந்துகள் பயன்படுத்தபடும். அவற்றில் ஒன்று டெக்ஸ்மெடெடொமிடின் என்பது .இந்த மருந்தினால் நோயாளிக்கு வலிநிவாரணம் , மிதமான தூக்கம் ஆகியவை ஏற்படும். இதனால் வலி மற்றும் தூக்கம் ஆகியவற்றிற்கு பயன்படுத்தபடும் மற்ற மருந்துகளின் அளவு குறைக்கபடும் . அவ்வாறு குறைக்கபடும் பொழுது அவற்றினால் ஏற்படும் பக்கவிளைவுகளும் குறையும். . அதனால் டெக்ஸ்மெடெடொமிடின் மருந்தை இருவேறு அளவில் பயன்படுத்தும்பொழுது மற்ற மருந்துகளின் அளவு தேவையை குறிப்பெடுத்த பின்னர் ஆராய்ச்சி செய்யபடும் .

ஆய்வு முறை :

இதில் நீங்கள் இரு குழுக்களாக பிரிக்கபடுவீர்கள் . ஒரு குழுவிர்க்கு 0.2 மைக்ரோக்ரம் / கிலோ / மணி அளவில் டெக்ஸ்மேடெடொமிடின் மற்றொரு குழுவிர்க்கு 0.6 மைக்ரோக்ரம் / கிலோ / மணி அளவில் டெக்ஸமெடெடொமிடின் கொடுக்கபடும் . இரத்தநானதில் மருந்து கொடுக்கும்பொழுது அதனால் ஏற்படும் இதய துடிப்பு , இரத்த அழுத்தம் மாற்றம் , மற்ற மயக்க மருந்துகளின் பயன்பாடு ஆகியவை குறிப்பெடுக்கபடும்.

உண்டாகக்கூடிய இடர்கள் :

இந்த ஆய்வின் பொழுது பயன்படுத்தபடும் டெக்ஸமெடெடொமிடின் மருந்தினால் , இதய துடிப்பு மற்றும் இரத்தஅழுத்தம் குறையக்கூடிய வாய்ப்புகள் உண்டு .

ஆய்வில் உங்கள் உரிமைகள் :

உங்கள் மருத்துவ பதிவேடுகள் அந்தரமாக வைத்துக்கொள்ளப்படும்இந்த .

ஆய்வின்முடிவுகள் அறிவியல் பத்திரிக்கைகளில் வெளியிடப் படலாம்ஆனால்.நீங்கள்.அடையாளம் காட்டப்பட மாட்டீர்கள் . இந்த ஆய்வில் பங்கேற்பது தன்னிச்சையானது மற்றும் காரணங்கள் எதுவும் கூறாமலேயே நீங்கள் எப்போது வேண்டுமென்றாலும் விலகிக் கொள்ளலாம்ஏதேனும் பக்க .

விளைவுகள் ஏற்பட்டால் முழு சிகிச்சையும். மருத்துவக்கு ழுவினரால். உடனடியாக வழங்கப்படும்

நோயாளியின் கையொப்பம்

நாள்

இடது பெருவிரல் ரேகை

MASTER CHART

SL No.	Group	Date	Name	IP No.	Age	Sex	Weight (kg)	Surgery	ASA	Duration of Surgery (mins)	Dexmedetomidine infusion (ug/kg/hr)	Induction
1	B	11/18/2013	Lalitha	38938	28	F	63	Lap . Cholecystectomy	I	90	0.6	Propofol 100 mg/Fentanyl 60 ug/Atracurium 25 mg
2	A	11/21/2013	Nazina Begum	37986	27	F	62	Lap . Cholecystectomy	I	80	0.2	Propofol 100 mg/Fentanyl 60 ug/Atracurium 25 mg
3	A	11/21/2013	Anbarasan	39731	23	M	71	Lap . Cholecystectomy	I	80	0.2	Propofol 100 mg/Fentanyl 70 ug/Atracurium 25 mg
4	A	11/22/2013	Ilakkiya	37021	22	F	59	Lap . Cholecystectomy	I	100	0.2	Propofol 100 mg/Fentanyl 60 ug/Atracurium 25 mg
5	B	11/29/2013	Maheswari	Page | 140784	42	F	61	Lap . Cholecystectomy	I	70	0.6	Propofol 100 mg/Fentanyl 60 ug/Atracurium 25 mg
6	B	1/7/2014	Saravana kumar	1400619	17	M	46	Lap . Cholecystectomy	I	120	0.6	Propofol 100 mg/Fentanyl 50 ug/Atracurium 25 mg
7	A	1/9/2013	Lingaraj	1401028	20	M	51	Lap . Cholecystectomy	I	110	0.2	Propofol 100 mg /Fentanyl 50 ug/Atracurium 25 mg
8	B	1/10/2014	Radha	1400803	42	F	61	Lap . Cholecystectomy	I	110	0.6	Propofol 120 mg/Fentanyl 60 ug/Atracurium 30mg
9	A	1/10/2014	Roja	1401803	25	F	52	Lap . Cholecystectomy	I	70	0.2	Propofol 100 mg/ Fentanyl 50 ug/Atracurium 25 mg
10	A	1/11/2014	Nazima	1400863	32	F	61	Lap . Cholecystectomy	I	80	0.2	Propofol 100 mg/Fentanyl 60 ug/Atracurium 25 mg
11	A	1/21/2014	Mukesh	1402523	20	M	62	Lap . Cholecystectomy	I	90	0.2	Propofol 120 mg/Fentanyl 60 ug/Atracurium 30mg
12	A	1/21/2014	Hemalatha	1402186	19	F	59	Lap . Cholecystectomy	I	70	0.2	Propofol 120 mg/Fentanyl 60 ug/Atracurium 30mg
13	B	1/24/2014	Regina	132412	32	F	52	Lap . Cholecystectomy	I	120	0.6	Propofol 100 mg/Fentanyl 50 ug/Atracurium 25 mg
14	A	1/29/2014	Elumalai	1403497	47	M	61	Lap . Cholecystectomy	I	110	0.2	Propofol 120 mg/Fentanyl 60 ug/Atracurium 30mg
15	A	2/3/2014	Mariraj	1404479	47	M	75	Lap . Cholecystectomy	I	190	0.2	Propofol 150 mg/Fentanyl 75ug/Atracurium 40 mg
16	B	2/6/2014	Revathy	140556	18	F	48	Lap . Cholecystectomy	I	90	0.6	Propofol 100 mg/ Fentanyl 50 ug/Atracurium 25 mg
17	A	2/12/2014	Selvi	1402843	50	F	51	Lap . Cholecystectomy	I	80	0.2	Propofol 100 mg/Fentanyl 60 ug/Atracurium 25 mg
18	A	2/19/2014	Tamaraiselvi	1405205	42	F	59	Lap . Cholecystectomy	I	110	0.2	Propofol 120 mg/Fentanyl 60 ug/Atracurium 30mg
19	A	2/20/2014	Devika	1407001	36	F	61	Lap . Cholecystectomy	I	120	0.2	Propofol 120 mg/Fentanyl 60ug/Atracurium 30mg
20	B	2/22/2014	Vignesh	8496	23	M	62	Lap . Cholecystectomy	I	80	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
21	B	2/27/2014	Annapoorani	8393	50	F	61	Lap . Cholecystectomy	I	90	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg

SL No.	Group	Date	Name	IP No.	Age	Sex	Weight (kg)	Surgery	ASA	Duration of Surgery (mins)	Dexmedetomidine infusion (ug/kg/hr)	Induction
22	B	2/28/2014	Jainu	7917	23	F	52	Lap . Cholecystectomy	I	90	0.6	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
23	A	3/4/2014	Yamuna	10100	20	F	49	Lap . Cholecystectomy	I	80	0.2	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
24	A	3/9/2014	Jagadha	10894	50	F	51	Lap . Cholecystectomy	I	100	0.2	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
25	A	3/10/2014	Dharani	21192	30	F	59	Lap . Cholecystectomy	I	80	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
26	A	5/7/2014	Anusha	21347	19	F	52	Lap . Cholecystectomy	I	90	0.2	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
27	B	5/9/2014	Selvi	21388	50	F	61	Lap . Cholecystectomy	I	130	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
28	B	5/10/2014	Sindhu	23674	15	F	51	Lap . Cholecystectomy	I	90	0.6	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
29	B	3/11/2014	Balakrishnan	8925	50	M	73	Lap . Cholecystectomy	I	90	0.6	Propofol 150 mg/Fentanyl 75ug/Atracurium 40 mg
30	A	5/13/2014	Thasriya nasllen	21456	45	F	59	Lap . Cholecystectomy	I	100	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
31	B	5/17/2014	Sathya	21763	30	F	62	Lap . Cholecystectomy	I	110	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
32	**B**	**5/20/2014**	**Uma**	**2447**	**45**	**F**	**58**	**Lap . Cholecystectomy**	**I**	**100**	**0.6**	**Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg**
33	A	5/19/2014	Deepa	23498	30	F	63	Lap . Cholecystectomy	I	90	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
34	A	5/21/2014	Selvi	19859	30	F	60	Lap . Cholecystectomy	I	90	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
35	B	2/25/2014	Sampoorna	23604	36	F	62	Lap . Cholecystectomy	I	90	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
36	B	2/23/2014	Geetha	20849	24	F	58	Lap . Cholecystectomy	I	90	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
37	A	2/11/2014	Pankajam	19201	36	F	64	Lap . Cholecystectomy	I	100	0.2	Propofol 130mg/Fentanyl 65ug/Atracurium 35 mg
38	A	2/6/2014	Rathinam	25192	42	F	59	Lap . Cholecystectomy	I	100	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
39	B	1/22/2014	Rajni	27243	29	F	62	Lap . Cholecystectomy	I	110	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
40	A	1/28/2014	Seetha	23491	34	F	57	Lap . Cholecystectomy	I	100	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
41	B	5/22/2014	Govindammal	25314	45	F	61	Lap . Cholecystectomy	I	110	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
42	A	3/5/2014	Ponmudi	4223	39	F	52	Lap . Cholecystectomy	I	80	0.2	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
43	B	5/15/2014	Kuppam	8501	45	M	69	Lap . Cholecystectomy	I	110	0.6	Propofol 140mg/Fentanyl 70ug/Atracurium 40mg

SL No.	Group	Date	Name	IP No.	Age	Sex	Weight (kg)	Surgery	ASA	Duration of Surgery (mins)	Dexmedetomidine infusion (ug/kg/hr)	Induction
44	A	3/11/2014	Kavitha	10815	44	F	52	Lap . Cholecystectomy	I	100	0.2	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
45	A	2/14/2014	Rani	23356	49	F	59	Lap . Cholecystectomy	I	100	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
46	B	5/23/2014	Vinothavalli	25714	45	F	62	Lap . Cholecystectomy	I	100	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
47	B	5/23/2014	Rekha	25434	33	F	58	Lap . Cholecystectomy	I	100	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
48	B	1/10/201774	Sundaram	22769	21	M	62	Lap . Cholecystectomy	I	100	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
49	B	1/22/2014	Hemalatha	21387	28	F	57	Lap . Cholecystectomy	I	80	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
50	A	1/24/2014	Suresh	26759	30	M	65	Lap . Cholecystectomy	I	100	0.2	Propofol 130mg/Fentanyl 65ug/Atracurium 35 mg
51	A	2/2/2014	Sarathy	24959	36	M	62	Lap . Cholecystectomy	I	110	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
52	A	2/21/2012	Govindammal	23408	40	F	57	Lap . Cholecystectomy	I	90	0.2	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
53	B	1/29/2014	Ramya	24351	42	F	52	Lap . Cholecystectomy	I	110	0.6	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
54	B	1/6/2014	Deepa	28421	32	F	61	Lap . Cholecystectomy	I	120	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
55	B	5/15/2014	Padma	21321	39	F	59	Lap . Cholecystectomy	I	100	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
56	B	5/11/2014	Charumathi	26543	43	F	63	Lap . Cholecystectomy	I	100	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
57	B	2/23/2014	Ganesh	28501	28	M	68	Lap . Cholecystectomy	I	80	0.6	Propofol 140mg/Fentanyl 70ug/Atracurium 40mg
58	B	5/29/2014	Sasikala	27835	23	F	52	Lap . Cholecystectomy	I	110	0.6	Propofol 100mg/Fentantyl 50ug/Atracurium 25mg
59	B	5/31/2014	Selvi	28123	27	F	59	Lap . Cholecystectomy	I	110	0.6	Propofol 120mg/Fentanyl 60ug/Atracurium 30 mg
60	A	6/1/2014	Menaka	26444	50	F	51	Lap . Cholecystectomy	I	80	0.2	Propofol 100 mg/Fentanyl 60 ug/Atracurium 25 mg

SL No.	Group	Date	Name	HEART RATE																											
				HR BL	HR Preinduction	HR Induction	HR Intubation	HR 1 min	HR 5 min	HR 10 min	HR 15 min	HR 20 min	HR 25 min	HR 30 min	HR 40 min	HR 50 min	HR 60 min	HR 70 min	HR 80 min	HR 90 min	HR 100 min	HR 110 min	HR120 min	HR 130 min	HR 140 min	HR 150 min	HR 160 min	HR 170 min	HR 180 min	HR 190 min	
1	B	11/18/2013	Lalitha	101	95	77	93	81	76	66	63	61	61	60	64	69	75	74	81	82											
2	A	11/21/2013	Nazina Begum	84	80	78	110	91	83	85	80	83	84	86	80	70	83	72	75												
3	A	11/21/2013	Anbarasan	84	80	78	110	91	83	80	81	85	82	86	80	70	83	72	80												
4	A	11/22/2013	Ilakkiya	95	90	84	113	94	91	81	81	82	80	80	79	81	76	71	70	82	84										
5	B	11/29/2013	Maheswari	97	99	106	96	95	90	72	65	65	64	62	63	63	62	68													
6	B	1/7/2014	Saravana kumar	69	78	74	88	76	69	59	64	65	64	62	60	58	58	56	56	59	52	50	73								
7	A	1/9/2013	Lingaraj	68	76	78	81	83	85	74	75	81	71	75	75	79	84	69	72	74	69	77									
8	B	1/10/2014	Radha	74	81	106	96	69	63	59	73	73	81	81	79	83	81	83	76	72	71	74	75	76							
9	A	1/10/2014	Roja	84	84	94	99	97	83	90	98	78	79	83	75	68	76	74													
10	A	1/11/2014	Nazima	94	87	82	94	96	97	82	85	86	84	82	79	75	72	77	88	102											
11	A	1/21/2014	Mukesh	68	68	77	111	92	62	77	77	55	54	51	56	56	56	56	75	76											
12	A	1/21/2014	Hemalatha	80	72	66	97	80	82	75	72	72	76	74	70	69	68	71													
13	B	1/24/2014	Regina	92	93	105	111	102	107	106	107	107	102	101	102	99	96	101	95	92	98	88	91								
14	A	1/29/2014	Elumalai	77	79	81	115	93	91	87	87	72	65	68	67	65	72	68	70	66	77	72									
15	A	2/3/2014	Mariraj	71	75	71	87	74	65	61	61	56	52	53	61	52	58	55	56	56	60	64	66	61	60	62	59	59	60	56	
16	B	2/6/2014	Revathy	77	88	101	106	113	92	96	99	92	95	92	85	84	81	82	78	71											
17	A	2/12/2014	Selvi	69	66	70	93	75	54	50	58	62	64	63	63	63	61	64	64												
18	A	2/19/2014	Tamaraiselvi	86	80	70	96	75	75	71	65	64	71	75	64	69	72	71	79	72	70										
19	A	2/20/2014	Devika	74	78	89	84	71	65	63	59	66	60	62	58	74	98	66	62	63	57	63	74	76							
20	B	2/22/2014	Vignesh	72	71	70	74	73	70	68	64	71	61	64	68	63	62	64	68	64											
21	B	2/27/2014	Annapoorani	108	92	95	96	80	89	82	80	88	80	82	83	79	80	83	85	78											
22	B	2/28/2014	Jainu	86	84	99	99	106	95	96	85	87	84	94	87	97	90	87	78	81											
23	A	3/4/2014	Yamuna	99	104	98	104	101	95	91	102	100	99	91	95	86	84	72	78												
24	A	3/9/2014	Jagadha	94	106	101	104	98	92	80	76	71	68	69	71	62	63	60	68												
25	A	3/10/2014	Dharani	88	90	90	82	83	74	74	72	75	79	92	98	67	65	66	69	65											
26	A	5/7/2014	Anusha	109	103	110	126	106	106	88	84	86	110	106	92	89	92	90	86	84											
27	B	5/9/2014	Selvi	98	96	86	92	83	81	78	75	88	85	83	82	78	76	93	72	73	70	70	70	70							
28	B	5/10/2014	Sindhu	79	84	72	110	96	88	77	66	72	74	72	72	73	72	75	84	86											
29	B	3/11/2014	Balakrishnan	65	64	68	74	73	63	67	71	67	65	69	68	63	65	61	65	69											
30	A	5/13/2014	Thasriya nasllen	100	90	92	89	76	74	68	58	58	56	60	59	65	60	65	70	86	85										
31	B	5/17/2014	Sathya	110	98	92	112	102	79	70	68	76	70	71	85	80	74	77	80	90	81	81									
32	B	5/20/2014	Uma	110	118	112	116	89	81	89	95	96	89	80	80	78	91	90	84	80	86										
33	A	5/19/2014	Deepa	108	102	110	125	106	105	88	83	86	110	106	92	87	92	90	86	84											
34	A	5/21/2014	Selvi	94	87	86	95	96	97	75	82	85	86	84	82	78	76	73	78	89											

SL No.	Group	Date	Name	HEART RATE																											
				HR BL	HR Preinduction	HR Induction	HR Intubation	HR 1 min	HR 5 min	HR 10 min	HR 15 min	HR 20 min	HR 25 min	HR 30 min	HR 40 min	HR 50 min	HR 60 min	HR 70 min	HR 80 min	HR 90 min	HR 100 min	HR 110 min	HR120 min	HR 130 min	HR 140 min	HR 150 min	HR 160 min	HR 170 min	HR 180 min	HR 190 min	
35	B	2/25/2014	Sampoorna	110	98	97	98	82	91	84	82	90	82	84	85	81	82	85	87	80											
36	B	2/23/2014	Geetha	112	100	99	100	84	93	86	84	92	84	86	87	83	84	89	82	84											
37	A	2/11/2014	Pankajam	95	88	85	95	97	98	76	83	86	87	85	83	80	76	73	78	89	101										
38	A	2/6/2014	Rathinam	94	87	84	94	96	97	75	82	85	86	84	82	79	75	72	77	88	100										
39	B	1/22/2014	Rajni	92	90	105	110	101	106	113	105	106	106	101	110	98	95	100	94	91	99	87									
40	A	1/28/2014	Seetha	74	79	81	115	93	91	87	87	72	65	68	67	65	72	68	70	71	66	77	70								
41	B	5/22/2014	Govindammal	104	95	98	95	85	84	85	80	80	80	84	85	74	70	70	70	70	71	75									
42	A	3/5/2014	Ponmudi	98	103	107	103	100	94	90	101	99	98	90	94	85	83	76	77												
43	B	5/15/2014	Kuppam	103	94	97	94	84	83	84	79	79	79	83	84	73	69	69	69	69	70	74									
44	A	3/11/2014	Kavitha	95	107	102	105	99	93	81	77	72	71	72	69	70	72	63	64	61	59										
45	A	2/14/2014	Rani	69	67	70	93	75	64	60	68	62	69	63	63	61	64	64	64	63	65										
46	B	5/23/2014	Vinothavalli	120	105	108	110	100	98	95	92	98	101	104	89	79	84	82	85	80	85										
47	B	5/23/2014	Rekha	118	103	106	108	98	96	93	90	96	99	102	87	77	82	80	83	78	83										
48	B	1/10/2014	Sundaram	69	78	74	87	75	68	72	60	62	64	63	59	57	57	55	55	58	57										
49	B	1/22/2014	Hemalatha	81	73	67	98	81	83	76	73	73	77	75	71	70	69	72	72												
50	A	1/24/2014	Suresh	68	68	77	111	92	62	77	64	77	55	54	51	56	56	56	56	58	76										
51	A	2/2/2014	Sarathy	74	79	81	115	93	91	87	87	72	65	68	67	65	72	68	70	66	77	79									
52	A	2/21/2012	Govindammal	93	86	85	94	95	96	74	81	84	85	83	81	77	75	72	77	88											
53	B	1/29/2014	Ramya	92	90	104	110	101	106	113	105	106	106	101	100	101	98	95	100	94	91	97	87								
54	B	1/6/2014	Deepa	74	81	106	96	69	63	62	59	73	81	81	79	83	81	83	76	72	71	74	75	76							
55	B	5/15/2014	Padma	110	118	112	116	89	81	89	95	96	81	80	80	78	91	90	84	80	86										
56	B	5/11/2014	Charumathi	108	116	110	114	87	79	87	93	94	79	78	78	76	89	88	82	78	84										
57	B	2/23/2014	Ganesh	72	71	70	74	73	70	68	64	71	61	64	68	63	62	64	68	64											
58	B	5/29/2014	Sasikala	82	80	81	110	97	88	77	73	80	82	78	76	77	67	65	68	67	61	68									
59	B	5/31/2014	Selvi	100	98	90	80	70	65	62	67	68	65	72	69	66	75	65	63	69	65	64									
60	A	6/1/2014	Menaka	69	66	70	93	75	54	50	58	62	64	63	63	63	61	64	64												

SL No.	Group	Date	Name	SYSTOLIC BLOOD PRESSURE																														
				SBP Baseline	SBP Preinduction	SBP Induction	SBP Intubation	SBP 1min	SBP 5 min	SBP 10 min	SBP 15 min	SBP 20 min	SBP 25 min	SBP 30 min	SBP 40 min	SBP 50 min	SBP 60 min	SBP 70 min	SBP 80 min	SBP 90 min	SBP 100 min	SBP 110 min	SBP 120 min	SBP 130MIN	SBP140 min	SBP 150 min	SBP160 min	SBP170 min	SBP180 min	SBP190 min	SBP200 min	SBP 210 min
1	B	11/18/2013	Lalitha	126	121	114	124	114	97	92	90	91	88	90	84	93	94	96	106	108														
2	A	11/21/2013	Nazina Begum	134	130	126	138	126	117	125	126	129	129	128	125	127	128	124	124															
3	A	11/21/2013	Anbarasan	134	129	126	136	126	117	125	126	129	129	128	125	127	128	114	125															
4	A	11/22/2013	Ilakkiya	110	108	100	110	100	91	122	118	112	91	82	108	112	110	112	106	114	122													
5	B	11/29/2013	Maheswari	161	150	146	151	140	136	123	120	114	114	117	153	141	142	138																
6	B	1/7/2014	Saravana kumar	122	122	112	126	119	100	94	81	94	94	99	105	104	100	102	101	103	102	99	122											
7	A	1/9/2013	Lingaraj	121	122	108	130	121	110	118	123	124	123	126	128	119	119	119	121	115	117	118	114											
8	B	1/10/2014	Radha	134	134	114	148	120	108	102	154	161	153	151	154	146	152	141	134	139	142	132	110	122										
9	A	1/10/2014	Roja	112	117	113	134	117	101	100	119	136	137	135	125	116	119	114																
10	A	1/11/2014	Nazima	127	119	119	134	114	113	134	125	126	119	123	123	115	115	112	119	106														
11	A	1/21/2014	Mukesh	133	144	122	118	151	122	114	131	117	130	142	140	142	116	122	136	134	126													
12	A	1/21/2014	Hemalatha	123	124	114	134	121	114	95	95	115	124	126	125	118	117	128																
13	B	1/24/2014	Regina	134	134	117	143	132	111	127	130	138	145	136	132	130	131	127	130	102	121	124	124											
14	A	1/29/2014	Elumalai	144	146	123	165	142	117	106	108	100	131	120	153	135	139	135	150	147	142	140												
15	A	2/3/2014	Mariraj	122	117	97	109	100	107	104	104	112	104	94	89	93	95	95	97	100	101	103	110	101	103	99	102	98	95	104				
16	B	2/6/2014	Revathy	113	123	107	131	120	115	123	149	126	119	112	104	130	100	99	107	114														
17	A	2/12/2014	Selvi	124	123	105	140	105	88	97	103	126	127	112	11	114	101	107	119															
18	A	2/19/2014	Tamaraiselvi	126	122	89	140	98	85	96	87	93	107	161	146	151	121	142	113	133	116													
19	A	2/20/2014	Devika	121	126	112	124	112	100	99	96	100	141	130	123	132	148	135	122	118	108	121	113	128										
20	B	2/22/2014	Vignesh	123	113	84	104	95	89	99	96	107	112	117	118	115	111	105	108	114														
21	B	2/27/2014	Annapoorani	161	171	150	165	132	119	93	118	147	128	126	136	126	121	131	129	121														
22	B	2/28/2014	Jainu	126	123	104	127	129	105	103	105	94	93	111	118	117	106	111	101	119														
23	A	3/4/2014	Yamuna	134	135	105	134	113	106	105	125	128	127	121	120	108	109	107	118															
24	A	3/9/2014	Jagadha	140	144	124	151	136	99	119	114	115	113	154	123	121	127	118	117	115	121													
25	A	3/10/2014	Dharani	132	126	124	164	148	117	117	114	113	131	139	124	117	119	125	126	117														
26	A	5/7/2014	Anusha	119	119	94	125	106	99	122	108	112	153	151	138	118	117	119	118	117														
27	B	5/9/2014	Selvi	154	158	116	149	115	107	81	139	155	147	145	149	124	126	125	109	107	128	106	110	110										
28	B	5/10/2014	Sindhu	100	95	98	109	102	97	90	84	115	113	123	119	128	119	125	103	105														
29	B	3/11/2014	Balakrishnan	139	136	114	174	125	102	81	113	155	154	155	148	129	130	103	108	108														
30	A	5/13/2014	Thasriya nasllen	141	149	129	109	117	119	115	109	119	126	153	156	150	125	132	113	109	113													
31	B	5/17/2014	Sathya	140	144	124	144	145	124	99	117	123	133	133	132	130	129	123	124	124	125	124												
32	B	5/20/2014	Uma	150	160	140	160	151	109	100	124	140	151	152	156	156	145	139	135	134	136													

SL No.	Group	Date	Name	SYSTOLIC BLOOD PRESSURE																														
				SBP Baseline	SBP Preinduction	SBP Induction	SBP Intubation	SBP 1min	SBP 5 min	SBP 10 min	SBP 15 min	SBP 20 min	SBP 25 min	SBP 30 min	SBP 40 min	SBP 50 min	SBP 60 min	SBP 70 min	SBP 80 min	SBP 90 min	SBP 100 min	SBP 110 min	SBP 120 min	SBP 130MIN	SBP140 min	SBP 150 min	SBP160 min	SBP170 min	SBP180 min	SBP190 min	SBP200 min	SBP 210 min
33	A	5/19/2014	Deepa	118	117	93	124	105	98	121	107	111	152	151	138	117	116	119	117	118														
34	A	5/21/2014	Selvi	128	120	102	135	115	114	110	135	126	127	120	124	124	116	116	113	120														
35	B	2/25/2014	Sampoorna	159	169	148	165	130	117	91	116	145	126	124	134	124	119	129	127	119														
36	B	2/23/2014	Geetha	154	164	143	160	125	112	86	11	140	121	119	129	119	114	124	122	114														
37	A	2/11/2014	Pankajam	128	120	109	135	4	110	135	126	117	120	124	124	116	116	113	120	107														
38	A	2/6/2014	Rathinam	127	119	108	134	114	113	104	134	125	116	119	123	123	115	115	112	119	106													
39	B	1/22/2014	Rajni	134	134	117	143	132	111	127	130	138	145	136	132	130	131	127	130	112	121	124	129											
40	A	1/28/2014	Seetha	144	146	123	145	142	117	106	108	100	131	120	153	135	139	135	150	145	147	142	140											
41	B	5/22/2014	Govindammal	123	122	121	121	108	121	109	96	95	90	94	94	94	115	96	96	109	108	112												
42	A	3/5/2014	Ponmudi	133	134	104	133	112	105	104	124	127	126	120	119	107	108	106	117															
43	B	5/15/2014	Kuppam	122	121	120	120	107	120	108	95	94	89	93	93	93	114	95	95	108	107	111												
44	A	3/11/2014	Kavitha	141	145	125	150	137	100	120	115	116	114	154	124	122	128	119	118	116	122													
45	A	2/14/2014	Rani	124	123	105	143	105	105	145	103	126	127	112	111	114	101	107	119	111	120													
46	B	5/23/2014	Vinothavalli	155	152	107	150	119	98	98	122	128	129	132	127	125	125	119	117	115	117													
47	B	5/23/2014	Rekha	153	150	105	148	117	96	96	120	126	127	130	125	123	123	117	115	113	115													
48	B	1/10/2014	Sundaram	121	121	111	125	118	99	97	93	93	93	93	98	104	103	99	101	100	102	101												
49	B	1/22/2014	Hemalatha	123	124	11	134	121	114	95	95	115	124	126	125	118	117	128	127															
50	A	1/24/2014	Suresh	124	145	119	152	123	104	115	132	118	131	143	141	143	117	123	137	135	127													
51	A	2/2/2014	Sarathy	143	145	122	144	142	116	105	107	99	129	119	152	134	138	134	149	147	141	139												
52	A	2/21/2012	Govindammal	128	120	102	135	115	114	110	135	126	127	120	124	124	116	116	113	120														
53	B	1/29/2014	Ramya	135	135	118	144	133	112	128	131	139	146	137	133	131	132	129	131	113	122	125	130											
54	B	1/6/2014	Deepa	134	134	114	148	120	108	99	102	148		148	153	151	154	146	152	141	134	139	142	132	110	122							
55	B	5/15/2014	Padma	150	150	140	159	151	109	100	124	140	151	152	156	156	145	139	135	134	136													
56	B	5/11/2014	Charumathi	145	145	135	154	146	104	95	119	135	146	147	151	151	140	134	130	129	131													
57	B	2/23/2014	Ganesh	123	113	84	104	95	89	99	96	107	112	117	118	115	111	105	108	114														
58	B	5/29/2014	Sasikala	120	102	96	107	110	104	97	94	97	118	115	113	106	107	101	97	100	111	125												
59	B	5/31/2014	Selvi	125	118	102	121	105	101	121	122	131	124	127	128	120	138	114	123	114	116	113												
60	A	6/1/2014	Menaka	124	123	105	140	105	88	97	103	126	127	112	11	114	101	107	119															

SL No.	Group	Date	Name	DIASTOLIC BLOOD PRESSURE																													
				DBP Baseline	DBP Preinduction	DBP Induction	DBP Intubation	DBP 1 min	DBP 5 min	DBP 10 min	DBP 15 min	DBP 20 min	DBP 25 min	DBP 30 min	DBP 40 min	DBP 50 min	DBP 60 min	DBP 70 min	DBP 80 min	DBP 90 min	DBP 100 min	DBP110 min	DBP 120 min	DBP130 min	DBP140 min	DBP150 min	DBP160 min	DBP170 min	DBP180 min	DBP190 min	DBP200 min	DBP210 min
1	B	11/18/2013	Lalitha	82	80	77	86	81	61	62	67	63	63	63	53	69	69	67	71	74													
2	A	11/21/2013	Nazina Begum	86	85	83	93	83	76	86	85	83	88	89	89	91	90	66	71														
3	A	11/21/2013	Anbarasan	86	84	83	93	83	76	83	85	88	88	89	89	91	90	66	84														
4	A	11/22/2013	Ilakkiya	70	68	60	70	60	58	76	80	86	70	60	88	82	86	84	80	82	88												
5	B	11/29/2013	Maheswari	113	108	101	101	97	96	91	96	85	85	82	83	102	102	98															
6	B	1/7/2014	Saravana kumar	73	80	69	87	76	58	58	46	57	58	62	69	68	64	65	63	64	66	61	80										
7	A	1/9/2013	Lingaraj	78	75	64	86	79	70	74	82	90	90	92	88	77	78	75	81	74	71	79	79										
8	B	1/10/2014	Radha	92	91	89	92	86	72	71	110	118	111	114	112	112	111	105	100	100	95	96	75	77									
9	A	1/10/2014	Roja	79	79	83	98	78	72	71	91	102	102	103	94	86	89	84															
10	A	1/11/2014	Nazima	78	77	77	85	75	79	99	91	86	77	88	84	79	79	80	79	66													
11	A	1/21/2014	Mukesh	91	86	75	109	79	59	88	79	88	97	96	104	86	84	88	87	87													
12	A	1/21/2014	Hemalatha	79	76	70	93	76	71	55	53	75	86	97	86	80	80	83															
13	B	1/24/2014	Regina	92	89	72	94	84	73	87	100	102	106	102	93	95	93	84	88	58	71	69	88										
14	A	1/29/2014	Elumalai	88	102	85	11	101	84	76	81	71	91	89	113	115	111	131	113	108	103												
15	A	2/3/2014	Mariraj	82	82	63	75	73	77	72	73	78	72	61	57	63	63	63	65	68	67	70	73	69	67	66	66	65	62	71			
16	B	2/6/2014	Revathy	78	88	68	97	86	81	87	93	88	82	76	73	65	68	66	75	73													
17	A	2/12/2014	Selvi	78	77	68	90	66	51	60	66	85	84	74	73	77	64	74	79														
18	A	2/19/2014	Tamaraiselvi	83	82	54	102	61	50	60	52	58	64	107	97	101	84	100	83	79	81												
19	A	2/20/2014	Devika	81	80	70	85	70	63	61	60	66	116	88	83	92	104	92	81	79	68	82	76	81									
20	B	2/22/2014	Vignesh	84	79	58	74	59	58	62	68	73	76	80	81	82	78	70	69	70													
21	B	2/27/2014	Annapoorani	94	102	88	106	82	78	63	71	94	82	81	82	86	79	80	80	79													
22	B	2/28/2014	Jainu	82	73	56	92	84	64	61	60	57	59	79	86	80	68	68	64	81													
23	A	3/4/2014	Yamuna	93	83	73	90	71	67	69	86	88	88	82	80	74	74	74	77														
24	A	3/9/2014	Jagadha	91	95	91	103	91	72	84	85	85	83	117	86	88	97	81	83	84	86												
25	A	3/10/2014	Dharani	89	84	80	113	102	85	85	80	81	97	104	86	83	83	88	84	84													
26	A	5/7/2014	Anusha	82	82	56	77	70	63	84	73	73	114	103	98	80	86	76	80	86													
27	B	5/9/2014	Selvi	106	107	77	104	83	75	60	106	109	108	105	104	84	90	90	80	77	91	73	76	76									
28	B	5/10/2014	Sindhu	70	65	65	85	85	74	51	58	54	83	83	89	85	87	89	89	73	79												
29	B	3/11/2014	Balakrishnan	92	91	98	104	97	81	66	89	115	114	114	108	98	97	78	82	82													
30	A	5/13/2014	Thasriya nasllen	97	86	86	76	76	77	77	69	80	85	114	105	108	82	92	75	76	76												

SL No.	Group	Date	Name	DIASTOLIC BLOOD PRESSURE																														
				DBP Baseline	DBP Preinduction	DBP Induction	DBP Intubation	DBP 1 min	DBP 5 min	DBP 10 min	DBP 15 min	DBP 20 min	DBP 25 min	DBP 30 min	DBP 40 min	DBP 50 min	DBP 60 min	DBP 70 min	DBP 80 min	DBP 90 min	DBP 100 min	DBP110 min	DBP 120 min	DBP130 min	DBP140 min	DBP150 min	DBP160 min	DBP170 min	DBP180 min	DBP190 min	DBP200 min	DBP210 min
31	B	5/17/2014	Sathya	79	79	69	84	86	71	60	78	86	93	89	84	86	85	81	81	79	78	79												
32	B	5/20/2014	Uma	80	86	80	99	98	85	70	85	98	100	101	106	102	97	93	92	94	92													
33	A	5/19/2014	Deepa	81	81	55	76	69	62	83	72	72	112	102	97	79	85	75	79	85														
34	A	5/21/2014	Selvi	79	78	64	86	76	80	68	100	92	87	78	89	85	80	80	81	80														
35	B	2/25/2014	Sampoorna	94	102	88	102	82	78	63	71	94	82	81	82	86	79	80	80	79														
36	B	2/23/2014	Geetha	89	97	83	97	77	73	58	66	89	77	76	77	81	74	75	75	74														
37	A	2/11/2014	Pankajam	79	78	71	86	76	80	68	100	92	87	78	89	85	80	80	81	80	67													
38	A	2/6/2014	Rathinam	78	77	70	85	75	79	67	99	91	86	77	88	84	79	79	80	79	66													
39	B	1/22/2014	Rajni	91	88	71	93	83	72	86	99	101	105	101	92	94	92	83	87	58	70	68	87											
40	A	1/28/2014	Seetha	88	102	85	111	101	84	76	81	71	91	89	113	103	105	100	121	107	102	92	86											
41	B	5/22/2014	Govindammal	83	82	80	90	88	80	75	64	64	61	64	64	66	83	68	68	79	78	82												
42	A	3/5/2014	Ponmudi	92	82	72	89	70	66	68	85	87	87	81	79	73	73	73	76															
43	B	5/15/2014	Kuppam	82	81	79	89	87	79	74	63	63	60	63	63	65	82	67	67	78	77	81												
44	A	3/11/2014	Kavitha	90	94	90	91	90	71	83	84	84	82	116	85	87	96	80	82	83	85													
45	A	2/14/2014	Rani	98	77	68	92	66	68	114	66	85	84	74	73	77	64	74	79	73	80													
46	B	5/23/2014	Vinothavalli	89	89	70	96	79	70	70	88	92	92	93	91	90	90	86	84	83	84													
47	B	5/23/2014	Rekha	88	88	69	95	78	69	69	87	91	91	92	90	89	89	85	83	82	83													
48	B	1/10/2014	Sundaram	73	80	69	87	76	58	60	58	58	57	58	62	69	68	64	65	63	64	66												
49	B	1/22/2014	Hemalatha	78	75	69	92	75	70	52	54	74	85	96	85	79	79	82	82															
50	A	1/24/2014	Suresh	82	87	76	110	80	60	79	89	80	89	98	97	105	87	85	89	88	88													
51	A	2/2/2014	Sarathy	88	102	85	111	101	84	76	81	71	91	89	113	103	105	100	121	102	92	86												
52	A	2/21/2012	Govindammal	78	77	63	85	75	79	67	99	91	88	77	88	84	79	79	80	79														
53	B	1/29/2014	Ramya	91	88	71	93	83	72	86	99	101	105	105	101	92	94	92	83	87	67	70	68	87										
54	B	1/6/2014	Deepa	92	91	89	92	86	72	62	71	92	92	111	114	112	112	111	105	100	100	95	96	75	77									
55	B	5/15/2014	Padma	80	86	80	99	98	85	70	85	98	100	101	106	102	97	93	92	94	92													
56	B	5/11/2014	Charumathi	82	88	82	101	100	87	72	87	100	102	103	108	104	99	95	94	96	94													
57	B	2/23/2014	Ganesh	84	79	58	74	59	58	62	68	73	76	80	81	82	78	70	69	70														
58	B	5/29/2014	Sasikala	72	68	64	77	60	57	51	56	59	80	81	64	58	73	66	60	64	63	79												
59	B	5/31/2014	Selvi	79	74	62	81	65	62	88	95	90	95	96	91	87	93	77	86	74	80	80												
60	A	6/1/2014	Menaka	78	77	68	90	66	51	60	66	85	84	74	73	77	64	74	79															

SL No.	Group	Date	Name	MEAN ARTERIAL PRESSURE (mm Hg)																												
				MAP Baseline	MAP Preinduction	MAP Induction	MAP Intubation	MAP 1 min	MAP 5 min	MAP 10 min	MAP 15 min	MAP 20 min	MAP 25 min	MAP 30 min	MAP 40 min	MAP 50 min	MAP 60 min	MAP 70 min	MAP 80 min	MAP 90 min	MAP 100min	MAP 110 min	MAP 120 min	MAP 130 min	MAP140 min	MAP 150 min	MAP160 min	MAP170 min	MAP180 min	MAP 190 min	MAP 200 min	MAP 210 min
1	B	11/18/2013	Lalitha	97	95	91	99	93	73	72	72	75	73	71	77	77	70	71	83	85												
2	A	11/21/2013	Nazina Begum	102	99	97	107	99	90	98	97	97	102	102	101	103	103	95	94													
3	A	11/21/2013	Anbarasan	102	99	97	107	97	90	97	100	102	101	102	101	103	103	95	97													
4	A	11/22/2013	Ilakkiya	83	78	73	83	73	71	90	92	95	81	67	95	89	94	90	88	92	99											
5	B	11/29/2013	Maheswari	129	121	116	116	111	109	102	100	95	95	93	94	119	112	113														
6	B	1/7/2014	Saravana kumar	86	93	82	102	89	72	70	58	69	72	74	61	80	77	72	76	77	78	74	92									
7	A	1/9/2013	Lingaraj	89	87	75	95	83	78	96	100	81	103	101	92	93	86	96	87	87	69	68										
8	B	1/10/2014	Radha	108	107	98	108	97	83	81	132	122	124	123	123	124	115	113	111	108	108	83										
9	A	1/10/2014	Roja	89	91	93	110	92	82	81	99	114	114	115	105	96	99															
10	A	1/11/2014	Nazima	92	88	88	98	89	91	102	102	99	89	97	96	91	91	90	94	80												
11	A	1/21/2014	Mukesh	104	104	86	123	92	70	101	91	101	110	108	115	98	95	101	101													
12	A	1/21/2014	Hemalatha	93	92	86	108	91	85	67	63	86	99	107	97	91	92	98														
13	B	1/24/2014	Regina	104	103	89	106	100	100	99	115	112	118	112	105	100	100	96	101	70	86	84	101									
14	A	1/29/2014	Elumalai	105	125	94	128	112	95	86	90	81	101	101	124	113	115	111	131	113	108	103										
15	A	2/3/2014	Mariraj	95	94	75	86	81	87	83	83	90	82	72	68	74	74	73	75	79	77	81	83	80	79	77	77	77	72	82		
16	B	2/6/2014	Revathy	89	100	77	108	96	93	99	109	102	96	89	84	77	80	77	85	85												
17	A	2/12/2014	Selvi	92	91	80	113	79	64	71	78	100	97	86	86	89	76	85	93													
18	A	2/19/2014	Tamaraiselvi	96	95	64	119	71	61	72	63	68	86	125	116	118	96	115	94	100	91											
19	A	2/20/2014	Devika	93	92	83	98	83	73	73	71	77	128	98	93	105	117	106	94	90	81	92	87	94								
20	B	2/22/2014	Vignesh	96	92	67	85	70	68	74	77	84	87	91	93	93	89	83	83	84												
21	B	2/27/2014	Annapoorani	118	141	114	117	102	92	72	90	118	103	98	100	102	92	98	95	92												
22	B	2/28/2014	Jainu	92	88	70	104	96	77	74	73	69	70	91	97	91	81	82	80	94												
23	A	3/4/2014	Yamuna	106	96	86	104	85	80	81	99	101	101	95	93	85	86	85	90													
24	A	3/9/2014	Jagadha	107	111	102	118	105	81	96	95	95	93	132	98	99	107	93	94	94	98											
25	A	3/10/2014	Dharani	103	98	95	130	117	96	96	91	92	108	116	99	94	95	100	96	96												
26	A	5/7/2014	Anusha	94	94	69	93	82	75	97	85	88	127	121	111	93	97	93	93	97												
27	B	5/9/2014	Selvi	124	125	88	119	94	86	67	117	124	121	118	119	101	102	102	90	87	103	84	87	87								
28	B	5/10/2014	Sindhu	80	76	76	93	94	60	69	64	94	93	100	96	97	98	99	83	82												
29	B	3/11/2014	Balakrishnan	108	109	105	127	106	88	71	97	128	127	128	121	107	108	86	91	91												
30	A	5/13/2014	Thasriya nasllen	110	110	100	88	90	91	90	82	93	99	130	122	122	96	105	85	87	86											
31	B	5/17/2014	Sathya	99	100	87	103	106	89	73	91	98	106	104	100	101	100	95	95	96	95	96										

32	B	5/20/2014	Uma	102	110	101	118	116	76	81	98	102	117	118	123	120	113	108	106	107	107													
33	A	5/19/2014	Deepa	93	93	68	92	81	74	97	84	87	126	120	110	92	96	92	92	96														
34	A	5/21/2014	Selvi	93	88	76	99	90	92	82	103	103	100	90	98	97	92	92	91	95														
35	B	2/25/2014	Sampoorna	117	140	113	121	101	91	71	90	118	103	98	100	102	92	98	95	92														
36	B	2/23/2014	Geetha	111	134	107	115	95	85	65	84	112	97	92	94	96	86	92	89	86														
37	A	2/11/2014	Pankajam	93	89	84	99	90	92	82	103	103	100	90	98	97	92	92	91	95	81													
38	A	2/6/2014	Rathinam	92	88	83	98	89	91	81	102	102	99	89	97	96	91	91	90	94	80													
39	B	1/22/2014	Rajni	104	103	89	106	100	100	99	115	112	118	112	105	108	100	96	101	70	86	84	101											
40	A	1/28/2014	Seetha	105	125	94	128	112	95	86	90	81	101	101	124	113	115	111	131	119	113	108	103											
41	B	5/22/2014	Govindammal	96	95	94	104	95	94	86	75	74	71	74	74	75	94	77	77	89	88	92												
42	A	3/5/2014	Ponmudi	105	95	85	103	84	79	80	98	100	100	94	92	84	85	84	99															
43	B	5/15/2014	Kuppam	96	95	94	104	95	94	86	75	74	71	74	74	75	94	77	77	89	88	92												
44	A	3/11/2014	Kavitha	106	110	101	112	106	80	95	94	94	92	131	97	98	106	92	93	93	97													
45	A	2/14/2014	Rani	92	91	80	109	79	79	125	78	100	96	86	86	89	76	85	93	86	95													
46	B	5/23/2014	Vinothavalli	111	110	84	114	92	79	79	99	104	104	106	103	102	102	97	95	94	95													
47	B	5/23/2014	Rekha	110	109	83	113	91	78	78	98	103	103	105	102	101	101	96	94	93	94													
48	B	1/10/2014	Sundaram	86	93	82	102	89	72	73	70	70	69	72	74	61	80	77	77	76	77	78												
49	B	1/22/2014	Hemalatha	92	91	85	107	90	84	68	62	85	98	106	96	90	91	97	97															
50	A	1/24/2014	Suresh	95	105	87	124	93	71	91	102	92	102	111	109	116	99	96	102	102	102													
51	A	2/2/2014	Sarathy	105	125	94	128	112	95	86	90	81	101	101	124	113	115	111	131	113	108	103												
52	A	2/21/2012	Govindammal	92	87	75	98	89	91	81	102	102	99	89	97	96	91	91	90	94														
53	B	1/29/2014	Ramya	104	103	89	106	100	100	99	115	112	118	112	105	100	100	96	101	80	86	84	101											
54	B	1/6/2014	Deepa	108	107	98	108	97	83	73	81	108	108	122	124	123	123	124	115	113	111	108	108	83	93									
55	B	5/15/2014	Padma	102	114	101	122	116	76	81	98	102	117	118	123	120	113	108	106	107	107													
56	B	5/11/2014	Charumathi	101	113	102	123	117	77	82	99	103	118	119	124	121	114	109	107	108	108													
57	B	2/23/2014	Ganesh	96	91	67	85	70	68	74	77	84	87	91	93	93	89	83	83	84														
58	B	5/29/2014	Sasikala	88	80	75	88	77	69	65	67	72	93	93	88	78	85	78	74	79	76	94												
59	B	5/31/2014	Selvi	91	88	75	94	78	70	98	105	103	106	105	103	98	105	88	97	86	92	91												
60	A	6/1/2014	Menaka	92	91	80	113	79	64	71	78	100	97	86	86	89	76	85	93															

SL No.	Group	Date	Name	END TIDAL CO2																							
				ETCO2 Intubation	ETCO21 min	ETCO25 min	ETCO2 10 min	ETCO2 15 min	ETCO2 20 min	ETCO2 25 min	ETCO2 30 min	ETCO2 40 min	ETCO2 50 min	ETCO2 60 min	ETCO2 70 min	ETCO2 80 min	ETCO2 90 min	ETCO2 100 min	ETCO2 110 min	ETCO2 120 min	ETCO2 130 min	ETCO2 140 min	ETCO2 150 min	ETCO2 160 min	ETCO2 170 min	ETCO2 180 min	ETCO2 190 min
1	B	11/18/2013	Lalitha	39	31	37	38	37	38	39	38	37	39	39	38	37	38										
2	A	11/21/2013	Nazina Begum	38	37	38	30	37	38	39	38	37	38	39	38	38											
3	A	11/21/2013	Anbarasan	38	37	37	39	30	36	38	39	37	38	38	38	36											
4	A	11/22/2013	Ilakkiya	38	38	39	38	39	36	37	39	38	39	30	37	38	38	38									
5	B	11/29/2013	Maheswari	30	31	30	30	38	38	39	30	38	39	30	30												
6	B	1/7/2014	Saravana kumar	39	38	39	36	38	39	30	37	38	30	36	39	38	37	39	30								
7	A	1/9/2013	Lingaraj	33	30	38	37	38	39	30	33	35	33	31	39	30	31	30	39								
8	B	1/10/2014	Radha	38	39	37	37	38	37	38	37	38	39	37	38	35	37	38	38	39	28	28					
9	A	1/10/2014	Roja	38	36	35	37	32	35	34	36	35															
10	A	1/11/2014	Nazima	38	39	30	34	32	31	30	30	38	39	39	39												
11	A	1/21/2014	Mukesh	39	38	39	38	37	39	30	39	37	39	38	37	39											
12	A	1/21/2014	Hemalatha	35	33	34	34	33	35	31	32	34	37	39													
13	B	1/24/2014	Regina	38	36	35	39	36	37	38	34	33	34	37	38	36	38	37	38								
14	A	1/29/2014	Elumalai	34	30	30	38	35	36	34	38	36	37	36	37	38	38										
15	A	2/3/2014	Mariraj	33	39	37	30	30	31	30	39	31	36	33	31	30	36	35	35	34	36	34	34	35	30	27	29
16	B	2/6/2014	Revathy	30	31	32	37	34	37	30	34	37	35	39	38	39											
17	A	2/12/2014	Selvi	34	34	36	37	34	34	33	35	38	31	30	31												
18	A	2/19/2014	Tamaraiselvi	31	31	31	37	38	34	38	37	38	37	39	38	39	39	30									
19	A	2/20/2014	Devika	33	34	37	34	35	32	36	34	30	32	35	34	30	30	30	30	34							
20	B	2/22/2014	Vignesh	33	38	37	34	39	31	39	31	39	37	39	30	38											
21	B	2/27/2014	Annapoorani	39	36	35	36	34	36	39	38	32	39	38	39	38	39										
22	B	2/28/2014	Jainu	39	30	38	30	31	39	30	32	30	32	30	32	30	30	31	30								
23	A	3/4/2014	Yamuna	30	38	39	39	29	34	36	35	33	36	34	30												
24	A	3/9/2014	Jagadha	30	30	37	39	37	31	30	34	35	37	38	37	34	37										
25	A	3/10/2014	Dharani	30	38	30	37	37	33	39	37	30	37	38	37	38											
26	A	5/7/2014	Anusha	38	36	35	34	32	35	36	34	36	35	37	36	37											
27	B	5/9/2014	Selvi	38	38	37	39	36	34	35	35	36	38	37	38	39	30	36	37								
28	B	5/10/2014	Sindhu	30	39	36	34	38	35	36	35	38	35	38	34	35	34	35									
29	B	3/11/2014	Balakrishnan	30	39	38	35	36	31	30	33	31	30	35	35												
30	A	5/13/2014	Thasriya nasllen	38	37	36	38	39	35	34	38	39	38	37	39	38	35	34									
31	B	5/17/2014	Sathya	38	37	34	35	37	36	37	38	37	36	38	37	36	36										
32	B	5/20/2014	Uma	39	38	39	36	38	37	38	37	36	37	37	38	37	37										

SL No.	Group	Date	Name	END TIDAL CO2																								
				ETCO2 Intubation	ETCO21 min	ETCO25 min	ETCO2 10 min	ETCO2 15 min	ETCO2 20 min	ETCO2 25 min	ETCO2 30 min	ETCO2 40 min	ETCO2 50 min	ETCO2 60 min	ETCO2 70 min	ETCO2 80 min	ETCO2 90 min	ETCO2 100 min	ETCO2 110 min	ETCO2 120 min	ETCO2 130 min	ETCO2 140 min	ETCO2 150 min	ETCO2 160 min	ETCO2 170 min	ETCO2 180 min	ETCO2 190 min
33	A	5/19/2014	Deepa	38	36	35	34	32	35	35	36	37	36	37	37	36												
34	A	5/21/2014	Selvi	38	38	39	30	34	34	33	34	35	35	37	38	36												
35	B	2/25/2014	Sampoorna	39	36	35	36	34	36	30	38	32	39	38	39	38	39											
36	B	2/23/2014	Geetha	39	36	35	36	34	36	39	38	32	39	38	39	38												
37	A	2/11/2014	Pankajam	30	30	39	38	36	37	38	39	36	35	36	36	37	38											
38	A	2/6/2014	Rathinam	38	38	37	37	36	35	38	39	39	38	38	39	37	37											
39	B	1/22/2014	Rajni	38	36	35	39	36	37	38	34	33	34	37	38	36	38	37	38									
40	A	1/28/2014	Seetha	34	30	30	38	35	36	34	38	36	37	36	37	38	38	38										
41	B	5/22/2014	Govindammal	35	35	35	34	34	33	34	31	31	35	34	35	34	35	35	34	35								
42	A	3/5/2014	Ponmudi	30	38	39	39	34	35	33	35	34	30															
43	B	5/15/2014	Kuppam	34	33	34	32	30	38	39	30	32	31	30	37	38	37	36	35	39								
44	A	3/11/2014	Kavitha	39	37	34	37	31	30	34	35	37	38	37	34	37												
45	A	2/14/2014	Rani	34	34	36	37	34	34	33	35	33	35	33	31	30	31	30										
46	B	5/23/2014	Vinothavalli	30	30	37	30	39	31	30	39	38	38	38	37	38												
47	B	5/23/2014	Rekha	30	30	39	30	39	31	30	39	39	38	39	38	39	38											
48	B	1/10/2014	Sundaram	38	39	30	39	39	39	38	35	36	35	35	38	38	38	35	38	30								
49	B	1/22/2014	Hemalatha	35	34	33	34	33	35	31	32	34	37	39	38													
50	A	1/24/2014	Suresh	39	38	39	38	39	38	36	35	38	39	38	37	39	35											
51	A	2/2/2014	Sarathy	34	30	30	38	35	36	33	38	36	37	36	37	38	38	38										
52	A	2/21/2012	Govindammal	38	38	39	30	34	34	33	34	35	35	36	38													
53	B	1/29/2014	Ramya	38	36	35	34	39	36	37	38	37	39	37	36	38	39											
54	B	1/6/2014	Deepa	37	38	39	36	35	36	35	34	38	39	38	39	38	38	37	36	39								
55	B	5/15/2014	Padma	32	31	32	30	39	39	38	38	39	37	36	37	38	37											
56	B	5/11/2014	Charumathi	30	30	31	39	38	37	38	39	38	39	38	38	39	39											
57	B	2/23/2014	Ganesh	32	32	31	30	32	39	38	37	38	37	39	38													
58	B	5/29/2014	Sasikala	33	30	39	38	37	39	37	36	38	39	35	34	35	37	38										
59	B	5/31/2014	Selvi	31	30	30	30	31	38	39	38	39	30	37	38	37	39	30										
60	A	6/1/2014	Menaka	34	34	36	37	34	34	33	35	38	31	30	31													

SL No.	Group	Date	Name	DESFLURANE CONCENTRATION																								
				DES Intubation	DES 1 min	DES5 min	DES 10 min	DES 15 min	DES 20 min	DES 25 min	DES 30 min	DES 40 min	DES50 min	DES 60 min	DES 70 min	DES 80 min	DES 90 min	DES 100 min	DES 110 min	DES 120 min	DES 130 min	DES 140 min	DES 150 min	DES 160 min	DES 170 min	DES 180 min	DES 190 min
1	B	11/18/2013	Lalitha	3	3	3	0	3	3	3	3	0	3	3	3	Skin closure												
2	A	11/21/2013	Nazina Begum	3	3	3	3	3	3	3	3	3	3	3	Skin Closure													
3	A	11/21/2013	Anbarasan	3	3	3	3	3	3	3	3	3	3	3	Skin Closure													
4	A	11/22/2013	Ilakkiya	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure												
5	B	11/29/2013	Maheswari	3	3	3	3	3	3	0	3	3	3	Skin closure														
6	B	1/7/2014	Saravana kumar	3	3	3	3	0	3	3	3	3	3	3	3	3	Skin closure											
7	A	1/9/2013	Lingaraj	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
8	B	1/10/2014	Radha	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	skin closure								
9	A	1/10/2014	Roja	3	3	3	3	3	3	3	3	Skin closure																
10	A	1/11/2014	Nazima	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure												
11	A	1/21/2014	Mukesh	3	3	3	3	3	3	3	3	3	3	3	Skin Closure													
12	A	1/21/2014	Hemalatha	3	3	3	3	3	3	3	3	3	3	Skin closure														
13	B	1/24/2014	Regina	3	3	3	3	3	3	3	3	3	3	3	3	3	0	Skin closure										
14	A	1/29/2014	Elumalai	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure										
15	A	2/3/2014	Mariraj	3	3	3	3	3	3	3	3	0	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure		
16	B	2/6/2014	Revathy	3	3	3	3	3	3	3	3	3	3	3	Skin Closure													
17	A	2/12/2014	Selvi	3	3	3	3	3	3	3	3	3	3	3	Skin Closure													
18	A	2/19/2014	Tamaraiselvi	3	3	0	3	0	0	3	3	3	3	3	3	3	3	Skin closure										
19	A	2/20/2014	Devika	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	skin closure								
20	B	2/22/2014	Vignesh	3	3	0	3	3	3	3	3	3	3	3	Skin Closure													
21	B	2/27/2014	Annapoorani	3	3	3	0	3	3	3	3	3	3	3	3	Skin closure												
22	B	2/28/2014	Jainu	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure												

SL No.	Group	Date	Name	DESFLURANE CONCENTRATION																							
				DES Intubation	DES 1 min	DES5 min	DES 10 min	DES 15 min	DES 20 min	DES 25 min	DES 30 min	DES 40 min	DES50 min	DES 60 min	DES 70 min	DES 80 min	DES 90 min	DES 100 min	DES 110 min	DES 120 min	DES 130 min	DES 140 min	DES 150 min	DES 160 min	DES 170 min	DES 180 min	DES 190 min
23	A	3/4/2014	Yamuna	3	3	3	3	3	3	3	3	3	3	3	Skin Closure												
24	A	3/9/2014	Jagadha	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure										
25	A	3/10/2014	Dharani	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
26	A	5/7/2014	Anusha	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
27	B	5/9/2014	Selvi	3	3	3	0	3	3	3	3	3	3	3	3	3	0	3	3	skin closure							
28	B	5/10/2014	Sindhu	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
29	B	3/11/2014	Balakrishnan	3	3	3	0	3	3	3	3	3	3	3	3	Skin closure											
30	A	5/13/2014	Thasriya nasllen	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure										
31	B	5/17/2014	Sathya	3	3	3	0	3	3	3	3	3	3	3	3	3	3	Skin closure									
32	B	5/20/2014	Uma	3	3	0	3	3	3	3	3	3	3	3	3	3	Skin closure										
33	A	5/19/2014	Deepa	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
34	A	5/21/2014	Selvi	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
35	B	2/25/2014	Sampoorna	3	3	3	0	3	3	3	3	3	3	3	3	Skin closure											
36	B	2/23/2014	Geetha	3	3	3	0	3	3	3	3	3	3	3	3	Skin closure											
37	A	2/11/2014	Pankajam	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure										
38	A	2/6/2014	Rathinam	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure										
39	B	1/22/2014	Rajni	3	3	3	3	3	3	3	3	3	3	3	3	3	0	Skin closure									
40	A	1/28/2014	Seetha	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure									
41	B	5/22/2014	Govindammal	3	3	3	3	3	3	0	3	3	3	3	3	3	3	Skin closure									
42	A	3/5/2014	Ponmudi	3	3	3	3	3	3	3	3	3	3	3	Skin Closure												
43	B	5/15/2014	Kuppam	3	3	3	3	3	3	0	3	3	3	3	3	3	3	Skin closure									
44	A	3/11/2014	Kavitha	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure										

SL No.	Group	Date	Name	DESFLURANE CONCENTRATION																							
				DES Intubation	DES 1 min	DES5 min	DES 10 min	DES 15 min	DES 20 min	DES 25 min	DES 30 min	DES 40 min	DES50 min	DES 60 min	DES 70 min	DES 80 min	DES 90 min	DES 100 min	DES 110 min	DES 120 min	DES 130 min	DES 140 min	DES 150 min	DES 160 min	DES 170 min	DES 180 min	DES 190 min
45	A	2/14/2014	Rani	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure										
46	B	5/23/2014	Vinothavalli	3	3	0	0	3	3	3	3	3	3	3	3	3	Skin closure										
47	B	5/23/2014	Rekha	3	3	0	0	3	3	3	3	3	3	3	3	3	Skin closure										
48	B	1/10/2014	Sundaram	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure									
49	B	1/22/2014	Hemalatha	3	3	3	3	3	3	3	3	3	3	3	Skin Closure												
50	A	1/24/2014	Suresh	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
51	A	2/2/2014	Sarathy	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure								
52	A	2/21/2012	Govindammal	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure											
53	B	1/29/2014	Ramya	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure									
54	B	1/6/2014	Deepa	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure								
55	B	5/15/2014	Padma	3	3	0	3	3	3	3	3	3	3	3	3	3	Skin closure										
56	B	5/11/2014	Charumathi	3	3	0	3	3	3	3	3	3	3	3	3	3	Skin closure										
57	B	2/23/2014	Ganesh	3	3	0	3	3	3	3	3	3	3	3	Skin Closure												
58	B	5/29/2014	Sasikala	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure									
59	B	5/31/2014	Selvi	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Skin closure									
60	A	6/1/2014	Menaka	3	3	3	3	3	3	3	3	3	3	3	Skin Closure												

SL No.	Group	Date	Name	INJECTION FENTANYL																									
				FEN Intubation	FEN1 min	FEN 5 min	FEN Skin Incision	FEN 10 min	FEN 15 min	FEN 20 min	FEN 25 min	FEN 30 min	FEN 40 min	FEN50 min	FEN60 min	FEN70 min	FEN80 min	FEN90 min	FEN100 min	FEN110 min	FEN120 min	FEN130 min	FEN140 min	FEN150 min	FEN160 min	FEN170 min	FEN180 min	FEN190 min
1	B	11/18/2013	Lalitha																										
2	A	11/21/2013	Nazina Begum																										
3	A	11/21/2013	Anbarasan																										
4	A	11/22/2013	Ilakkiya																										
5	B	11/29/2013	Maheswari																										
6	B	1/7/2014	Saravana kumar																										
7	A	1/9/2013	Lingaraj																										
8	B	1/10/2014	Radha								30																		
9	A	1/10/2014	Roja									25																	
10	A	1/11/2014	Nazima																										
11	A	1/21/2014	Mukesh																										
12	A	1/21/2014	Hemalatha																										
13	B	1/24/2014	Regina																										
14	A	1/29/2014	Elumalai														30												
15	A	2/3/2014	Mariraj																										
16	B	2/6/2014	Revathy																										
17	A	2/12/2014	Selvi																										
18	A	2/19/2014	Tamaraiselvi									25		25															
19	A	2/20/2014	Devika												30														
20	B	2/22/2014	Vignesh																										
21	B	2/27/2014	Annapoorani																										
22	B	2/28/2014	Jainu																										
23	A	3/4/2014	Yamuna																										
24	A	3/9/2014	Jagadha									25																	
25	A	3/10/2014	Dharani		30																								
26	A	5/7/2014	Anusha								25	25																	
27	B	5/9/2014	Selvi																										
28	B	5/10/2014	Sindhu																										
29	B	3/11/2014	Balakrishnan																										
30	A	5/13/2014	Thasriya nasllen									30																	
31	B	5/17/2014	Sathya																										
32	B	5/20/2014	Uma																										

SL No.	Group	Date	Name	INJECTION FENTANYL																									
				FEN Intubation	FEN1 min	FEN 5 min	FEN Skin Incision	FEN 10 min	FEN 15 min	FEN 20 min	FEN 25 min	FEN 30 min	FEN 40 min	FEN50 min	FEN60 min	FEN70 min	FEN80 min	FEN90 min	FEN100 min	FEN110 min	FEN120 min	FEN130 min	FEN140 min	FEN150 min	FEN160 min	FEN170 min	FEN180 min	FEN190 min
33	A	5/19/2014	Deepa								30	30																	
34	A	5/21/2014	Selvi																										
35	B	2/25/2014	Sampoorna																										
36	B	2/23/2014	Geetha																										
37	A	2/11/2014	Pankajam																										
38	A	2/6/2014	Rathinam																										
39	B	1/22/2014	Rajni																										
40	A	1/28/2014	Seetha														30												
41	B	5/22/2014	Govindammal																										
42	A	3/5/2014	Ponmudi																										
43	B	5/15/2014	Kuppam																										
44	A	3/11/2014	Kavitha									25																	
45	A	2/14/2014	Rani					30																					
46	B	5/23/2014	Vinothavalli																										
47	B	5/23/2014	Rekha																										
48	B	1/10/2014	Sundaram																										
49	B	1/22/2014	Hemalatha																										
50	A	1/24/2014	Suresh										30																
51	A	2/2/2014	Sarathy																30										
52	A	2/21/2012	Govindammal																										
53	B	1/29/2014	Ramya																										
54	B	1/6/2014	Deepa																										
55	B	5/15/2014	Padma																										
56	B	5/11/2014	Charumathi																										
57	B	2/23/2014	Ganesh																										
58	B	5/29/2014	Sasikala																										
59	B	5/31/2014	Selvi																										
60	A	6/1/2014	Menaka																										

SL No.	Group	Date	Name	Fluid Bolus / Injection Ephedrine																										
				FL Intubation	FL1 min	FL 5 min	FL Skin Incision	FL 10 min	FL 15 min	FL 20 min	FL 25 min	FL 30 min	FL 40 min	FL 50 min	FL 60 min	FL 70 min	FL 80 min	FL 90 min	FL 100 min	FL 110 min	FL 120 min	FL 130 min	FL 140 min	FL 150 min	FL 160 min	FL 170 min	FL 180 min	FL190 min
1	B	11/18/2013	Lalitha					200 ml					200 ml																	
2	A	11/21/2013	Nazina Begum																											
3	A	11/21/2013	Anbarasan																											
4	A	11/22/2013	Ilakkiya																											
5	B	11/29/2013	Maheswari										200 ml																	
6	B	1/7/2014	Saravana kumar																											
7	A	1/9/2013	Lingaraj																											
8	B	1/10/2014	Radha																											
9	A	1/10/2014	Roja																											
10	A	1/11/2014	Nazima																											
11	A	1/21/2014	Mukesh																											
12	A	1/21/2014	Hemalatha																											
13	B	1/24/2014	Regina															200 ml												
14	A	1/29/2014	Elumalai																											
15	A	2/3/2014	Mariraj										200 ml																	
16	B	2/6/2014	Revathy																											
17	A	2/12/2014	Selvi																											
18	A	2/19/2014	Tamaraiselvi																											
19	A	2/20/2014	Devika																											
20	B	2/22/2014	Vignesh			200 ml																								
21	B	2/27/2014	Annapoorani					200 ml																						
22	B	2/28/2014	Jainu																											
23	A	3/4/2014	Yamuna																											
24	A	3/9/2014	Jagadha																											
25	A	3/10/2014	Dharani																											
26	A	5/7/2014	Anusha																											
27	B	5/9/2014	Selvi					200 ml										200 ml												
28	B	5/10/2014	Sindhu																											
29	B	3/11/2014	Balakrishnan					200 ml																						
30	A	5/13/2014	Thasriya nasllen																											
31	B	5/17/2014	Sathya					200 ml																						

SL No.	Group	Date	Name	Fluid Bolus / Injection Ephedrine																										
				FL Intubation	FL1 min	FL 5 min	FL Skin Incision	FL 10 min	FL 15 min	FL 20 min	FL 25 min	FL 30 min	FL 40 min	FL 50 min	FL 60 min	FL 70 min	FL 80 min	FL 90 min	FL 100 min	FL 110 min	FL 120 min	FL 130 min	FL 140 min	FL 150 min	FL 160 min	FL 170 min	FL 180 min	FL190 min
32	B	5/20/2014	Uma			200 ml																								
33	A	5/19/2014	Deepa																											
34	A	5/21/2014	Selvi																											
35	B	2/25/2014	Sampoorna					200 ml																						
36	B	2/23/2014	Geetha					200 ml																						
37	A	2/11/2014	Pankajam																											
38	A	2/6/2014	Rathinam																											
39	B	1/22/2014	Rajni															200 ml												
40	A	1/28/2014	Seetha																											
41	B	5/22/2014	Govindammal								200 ml																			
42	A	3/5/2014	Ponmudi																											
43	B	5/15/2014	Kuppam								200 ml																			
44	A	3/11/2014	Kavitha																											
45	A	2/14/2014	Rani																											
46	B	5/23/2014	Vinothavalli			200 ml		200 ml																						
47	B	5/23/2014	Rekha			200 ml		200 ml																						
48	B	1/10/2014	Sundaram																											
49	B	1/22/2014	Hemalatha																											
50	A	1/24/2014	Suresh																											
51	A	2/2/2014	Sarathy																											
52	A	2/21/2012	Govindammal																											
53	B	1/29/2014	Ramya																											
54	B	1/6/2014	Deepa																											
55	B	5/15/2014	Padma			200 ml																								
56	B	5/11/2014	Charumathi			200 ml																								
57	B	2/23/2014	Ganesh			200 ml																								
58	B	5/29/2014	Sasikala																											
59	B	5/31/2014	Selvi																											
60	A	6/1/2014	Menaka																											

SL No.	Group	Date	Name	Dexmedetomidine Infusion																										
				DEX Preinduction	DEX Induction	DEX Intubation	DEX 1 min	DEX 5 min	DEX 10 min	DEX 15 min	DEX 20 min	DEX 25 min	DEX 30 min	DEX 40 min	DEX 50 min	DEX 60 min	DEX 70 min	DEX 80 min	DEX 90 min	DEX 100 min	DEX 110 min	DEX 120 min	DEX 130 min	DEX 140 min	DEX 150 min	DEX 160 min	DEX 170 min	DEX 180 min	DEX 190 min
1	B	11/18/2013	Lalitha	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure												
2	A	11/21/2013	Nazina Begum	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped - skin closure													
3	A	11/21/2013	Anbarasan	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped - skin closure													
4	A	11/22/2013	Ilakkiya	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											
5	B	11/29/2013	Maheswari	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure														
6	B	1/7/2014	Saravana kumar	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure											
7	A	1/9/2013	Lingaraj	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											
8	B	1/10/2014	Radha	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure								
9	A	1/10/2014	Roja	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure																	
10	A	1/11/2014	Nazima	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure												
11	A	1/21/2014	Mukesh	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure												
12	A	1/21/2014	Hemalatha	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	stopped - skin closure														
13	B	1/24/2014	Regina	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure										
14	A	1/29/2014	Elumalai	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	stopped -skin closure										
15	A	2/3/2014	Mariraj	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure		
16	B	2/6/2014	Revathy	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure													
17	A	2/12/2014	Selvi	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped - skin closure	-	-	-										

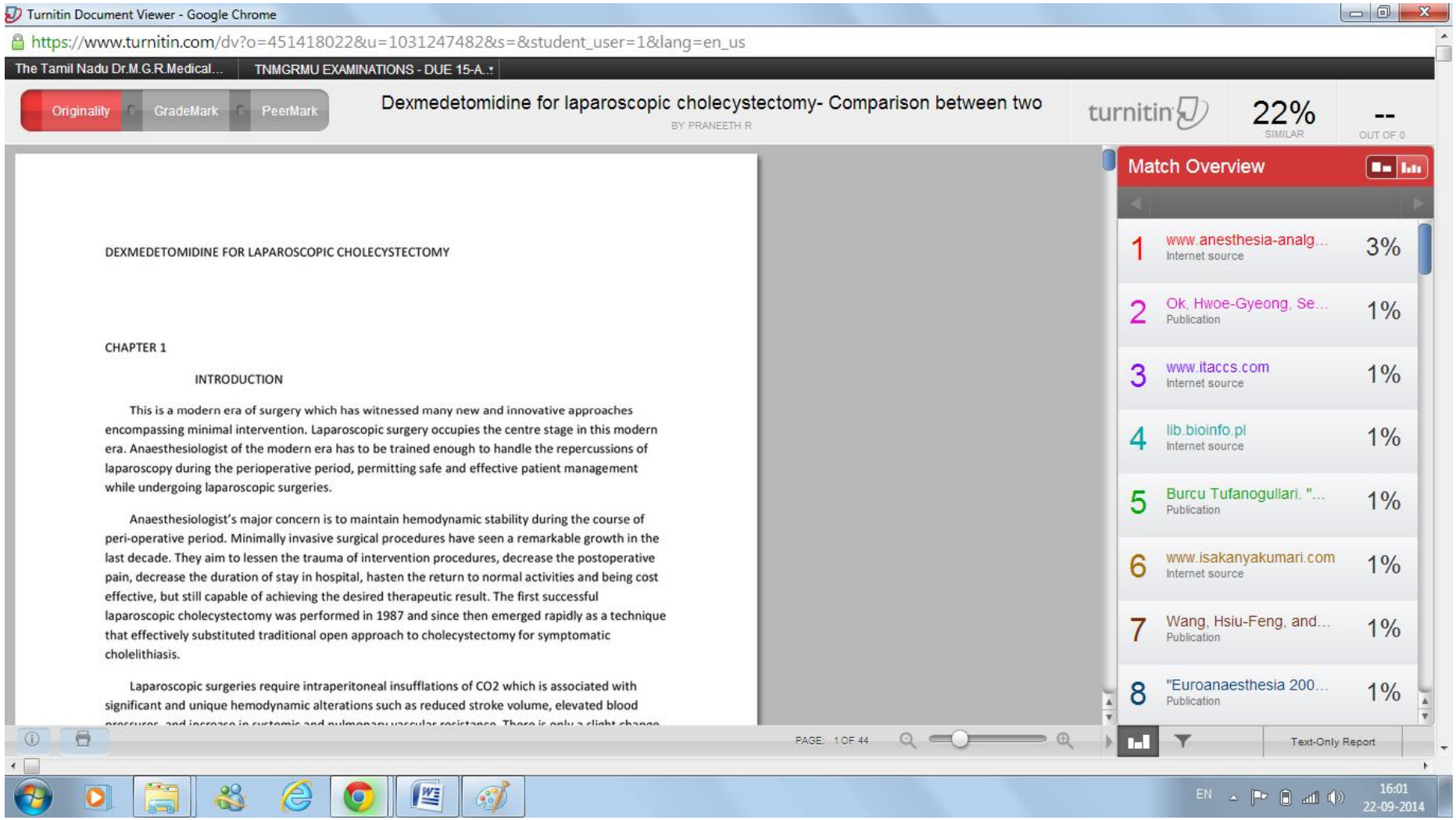
SL No.	Group	Date	Name	Dexmedetomidine Infusion																									
				DEX Preinduction	DEX Induction	DEX Intubation	DEX 1 min	DEX 5 min	DEX 10 min	DEX 15 min	DEX 20 min	DEX 25 min	DEX 30 min	DEX 40 min	DEX 50 min	DEX 60 min	DEX 70 min	DEX 80 min	DEX 90 min	DEX 100 min	DEX 110 min	DEX 120 min	DEX 130 min	DEX 140 min	DEX 150 min	DEX 160 min	DEX 170 min	DEX 180 min	DEX 190 min
18	A	2/19/2014	Tamaraiselvi	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
19	A	2/20/2014	Devika	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped - skin closure							
20	B	2/22/2014	Vignesh	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure												
21	B	2/27/2014	Annapoorani	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure											
22	B	2/28/2014	Jainu	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure											
23	A	3/4/2014	Yamuna	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped - skin closure												
24	A	3/9/2014	Jagadha	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
25	A	3/10/2014	Dharani	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											
26	A	5/7/2014	Anusha	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											
27	B	5/9/2014	Selvi	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure							
28	B	5/10/2014	Sindhu	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure											
29	B	3/11/2014	Balakrishnan	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure											
30	A	5/13/2014	Thasriya nasllen	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
31	B	5/17/2014	Sathya	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure									
32	B	5/20/2014	Uma	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure										
33	A	5/19/2014	Deepa	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											
34	A	5/21/2014	Selvi	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											

SL No.	Group	Date	Name	Dexmedetomidine Infusion																									
				DEX Preinduction	DEX Induction	DEX Intubation	DEX 1 min	DEX 5 min	DEX 10 min	DEX 15 min	DEX 20 min	DEX 25 min	DEX 30 min	DEX 40 min	DEX 50 min	DEX 60 min	DEX 70 min	DEX 80 min	DEX 90 min	DEX 100 min	DEX 110 min	DEX 120 min	DEX 130 min	DEX 140 min	DEX 150 min	DEX 160 min	DEX 170 min	DEX 180 min	DEX 190 min
35	B	2/25/2014	Sampoorna	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure											
36	B	2/23/2014	Geetha	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure											
37	A	2/11/2014	Pankajam	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
38	A	2/6/2014	Rathinam	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
39	B	1/22/2014	Rajni	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure									
40	A	1/28/2014	Seetha	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
41	B	5/22/2014	Govindammal	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure									
42	A	3/5/2014	Ponmudi	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped - skin closure												
43	B	5/15/2014	Kuppam	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure									
44	A	3/11/2014	Kavitha	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
45	A	2/14/2014	Rani	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure										
46	B	5/23/2014	Vinothavalli	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure										
47	B	5/23/2014	Rekha	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure										
48	B	1/10/2014	Sundaram	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure									
49	B	1/22/2014	Hemalatha	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure												
50	A	1/24/2014	Suresh	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											
51	A	2/2/2014	Sarathy	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure									

SL No.	Group	Date	Name	Dexmedetomidine Infusion																									
				DEX Preinduction	DEX Induction	DEX Intubation	DEX 1 min	DEX 5 min	DEX 10 min	DEX 15 min	DEX 20 min	DEX 25 min	DEX 30 min	DEX 40 min	DEX 50 min	DEX 60 min	DEX 70 min	DEX 80 min	DEX 90 min	DEX 100 min	DEX 110 min	DEX 120 min	DEX 130 min	DEX 140 min	DEX 150 min	DEX 160 min	DEX 170 min	DEX 180 min	DEX 190 min
52	A	2/21/2012	Govindammal	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped -skin closure											
53	B	1/29/2014	Ramya	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure									
54	B	1/6/2014	Deepa	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure								
55	B	5/15/2014	Padma	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure										
56	B	5/11/2014	Charumathi	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure										
57	B	2/23/2014	Ganesh	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped - skin closure												
58	B	5/29/2014	Sasikala	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure								
59	B	5/31/2014	Selvi	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	Stopped -skin closure								
60	A	6/1/2014	Menaka	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2	Stopped - skin closure	-	-	-									

SL No.	Group	Date	Name	Duration to Recovery (Ability to obey commands)	Duration to Tracheal Extubation (min)	Post extubation Sedation Score (RAMSAY)	Average Inspiratory Desflurane Concentration	Total Fentanyl	Hypotension episodes	Fentanyl bolus
1	B	11/18/2013	Lalitha	17	19	2	2.33	60	2	0
2	A	11/21/2013	Nazina Begum	23	25	2	2.65	60	0	0
3	A	11/21/2013	Anbarasan	21	23	2	2.65	70	0	0
4	A	11/22/2013	Ilakkiya	17	20	2	2.68	50	0	0
5	B	11/29/2013	Maheswari	17	19	2	2.4	60	1	0
6	B	1/7/2014	Saravana kumar	16	20	3	2.57	60	0	0
7	A	1/9/2013	Lingaraj	7	11	2	2.714	50	0	0
8	B	1/10/2014	Radha	20	24	2	2.78	90	0	1
9	A	1/10/2014	Roja	16	20	2	2.45	75	0	1
10	A	1/11/2014	Nazima	14	16	2	2.68	60	0	0
11	A	1/21/2014	Mukesh	18	22	2	2.65	60	0	0
12	A	1/21/2014	Hemalatha	9	13	2	2.6	60	0	0
13	B	1/24/2014	Regina	16	20	3	2.48	50	1	0
14	A	1/29/2014	Elumalai	11	15	2	2.74	80	0	1
15	A	2/3/2014	Mariraj	16	18	2	2.85	75	1	0
16	B	2/6/2014	Revathy	18	21	2	2.65	50	0	0
17	A	2/12/2014	Selvi	11	13	2	2.65	60	0	0
18	A	2/19/2014	Tamaraiselvi	12	15	2	2.35	100	0	2
19	A	2/20/2014	Devika	12	14	2	2.78	100	0	1
20	B	2/22/2014	Vignesh	17	20	2	2.47	60	1	0
21	B	2/27/2014	Annapoorani	18	22	3	2.53	60	1	0
22	B	2/28/2014	Jainu	18	22	3	2.68	50	0	0
23	A	3/4/2014	Yamuna	15	17	2	2.65	50	0	0
24	A	3/9/2014	Jagadha	16	19	2	2.71	75	0	1
25	A	3/10/2014	Dharani	15	19	2	2.68	90	0	1
26	A	5/7/2014	Anusha	14	18	2	2.68	100	0	2
27	B	5/9/2014	Selvi	18	21	2	2.44	60	2	0
28	B	5/10/2014	Sindhu	17	20	2	2.68	50	0	0
29	B	3/11/2014	Balakrishnan	18	20	2	2.53	75	1	0
30	A	5/13/2014	Thasriya nasllen	15	17	2	2.71	90	0	1
31	B	5/17/2014	Sathya	18	22	2	2.61	60	1	0
32	B	5/20/2014	Uma	16	19	3	2.57	60	1	0
33	A	5/19/2014	Deepa	15	18	2	2.68	120	0	2
34	A	5/21/2014	Selvi	14	18	2	2.68	60	0	0

SL No.	Group	Date	Name	Duration to Recovery (Ability to obey commands)	Duration to Tracheal Extubation (min)	Post extubation Sedation Score (RAMSAY)	Average Inspiratory Desflurane Concentration	Total Fentanyl	Hypotension episodes	Fentanyl bolus
35	B	2/25/2014	Sampoorna	17	21	2	2.53	60	1	0
36	B	2/23/2014	Geetha	18	22	2	2.53	60	1	0
37	A	2/11/2014	Pankajam	15	17	2	2.71	65	0	0
38	A	2/6/2014	Rathinam	14	16	2	2.71	60	0	0
39	B	1/22/2014	Rajni	16	19	2	2.48	60	1	0
40	A	1/28/2014	Seetha	14	17	2	2.74	90	0	1
41	B	5/22/2014	Govindammal	16	19	2	2.61	60	1	0
42	A	3/5/2014	Ponmudi	13	16	2	2.65	50	0	0
43	B	5/15/2014	Kuppam	15	19	2	2.61	65	1	0
44	A	3/11/2014	Kavitha	14	16	2	2.71	75	0	0
45	A	2/14/2014	Rani	15	17	2	2.71	90	0	1
46	B	5/23/2014	Vinothavalli	16	20	2	2.41	60	2	1
47	B	5/23/2014	Rekha	15	18	2	2.42	60	2	0
48	B	1/10/2014	Sundaram	16	19	2	2.74	60	0	0
49	B	1/22/2014	Hemalatha	18	22	2	2.65	60	0	0
50	A	1/24/2014	Suresh	15	17	2	2.68	90	0	1
51	A	2/2/2014	Sarathy	14	17	2	2.76	90	0	1
52	A	2/21/2012	Govindammal	13	17	2	2.68	60	0	0
53	B	1/29/2014	Ramya	16	20	2	2.74	50	0	0
54	B	1/6/2014	Deepa	17	21	2	2.76	60	0	0
55	B	5/15/2014	Padma	15	18	2	2.57	60	1	0
56	B	5/11/2014	Charumathi	16	19	2	2.56	60	1	0
57	B	2/23/2014	Ganesh	17	20	2	2.43	70	1	0
58	B	5/29/2014	Sasikala	18	20	2	2.74	50	0	0
59	B	5/31/2014	Selvi	16	19	2	2.74	60	0	0
60	A	6/1/2014	Menaka	11	13	2	2.65	60	0	0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CHAPTER 1
INTRODUCTION
This is a modern era of surgery which has witnessed many new and innovative approaches encompassing minimal intervention. Laparoscopic surgery occupies the centre stage in this modern era. Anaesthesiologist of the modern era has to be trained enough to handle the repercussions of laparoscopy during the perioperative period, permitting safe and effective patient management while undergoing laparoscopic surgeries.
Anaesthesiologist's major concern is to maintain hemodynamic stability during the course of peri-operative period. Minimally invasive surgical procedures have seen a remarkable growth in the last decade. They aim to lessen the trauma of intervention procedures, decrease the postoperative pain, decrease the duration of stay in hospital, hasten the return to normal activities and being cost effective, but still capable of achieving the desired therapeutic result. The first successful laparoscopic cholecystectomy was performed in 1987 and since then emerged rapidly as a technique that effectively substituted traditional open approach to cholecystectomy for symptomatic cholelithiasis.
Laparoscopic surgeries require intraperitoneal insufflations of CO2 which is associated with significant and unique hemodynamic alterations such as reduced stroke volume, elevated blood
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